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Original Articles and Clinical Cases.

MENTAL WANDERING.

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In addressing the Neurological Society it seemed well for me to select a topic not entirely within the strict limits of insanity, to which my work has chiefly been devoted; and again, as leisure time promised to be short, one inclined to something comparatively simple and of general interest.

The subject selected is "Mental Wandering," by which I mean subdelirious conditions, delirious states, and certain dream-modifications holding definite relations thereto. The one word "delirium" may be thought sufficient; but "delirium" has been used with a considerable variety of meanings; in some countries applied to many symptom-assemblages, or even definite clinical forms of insanity; in this country usually with a much more narrow significance, but still a wide one, though shorn of elements that properly belong to it, I think. By the title, "mental wandering," fuller explanation is saved, some contentious matter avoided, and one can still freely use the word "delirium" with the meaning more usually attached to it here.

As a rule, the study of mental symptoms, especially of the less severe, must be both objective and subjective; and,

like its congeners, mental wandering presents many features only known, or knowable, through the patient's self-observation. This last has obvious limitations. Nevertheless, some examples are especially suitable; the development of the subjective symptoms being in such degree and poise as to be unusually favourable to introspection, and distinctly recorded. The source of our knowledge of such cases must be the patient's account. And instead of dealing formally with the theme, it may be of more interest if we glance together at an example of this favourable kind which I have met, in the hope of finding some simple practical points and some broad truths concerning nervous and mental states, and of observing something of the passage from normal mind to abnormal.

The patient was a medical man, who, for the present purpose, will be named "A." His only mental wandering was on the occasion referred to, and a result of typhoid fever; his attacks of parotitis, measles, scarlet fever, influenza (twice), besides other febrile maladies, and brain concussion, were all free from any noteworthy mental wandering.

A number of others were infected at the same time as "A." All of them were said to have died. Thus we already meet with a practical point; the support yielded to the old view as to the existence of strong contrasts in the virulence of the infection in different outbreaks or from different sources.

The first slight deviation from health came on the night following the expiration of fourteen days from the taking of the virus, sleep on that night being defective and uneasy, and next morning a light swimming head-sensation was felt, and at first aroused ideà of oncoming frontal-sinus, and nasal catarrh. For several days work was continued, and even an extra piece of night-work was done on the third night after the appearance of those symptoms. During these days and the next, malaise, weakness, and slight chilliness presaged the coming trouble; and there was something of that grey, tallowy, and stricken look sometimes seen in infections after the close of incubation and with the

onset. On the fourth day the patient could not leave the house, and had raised temperature, but wrote letters, and suspected typhoid fever; and on the fifth day felt sure of it, and was so ill that he beat a retreat to bed, with high temperature, some headache, mental confusion, and budding delirium.

To-night we must not delay with the general clinical symptoms. An abstract of these forms an appendix, enabling some comparison between the general and the mental states.

But it must be mentioned now that, in comparison with the febrile movement as measured by body-temperature, the toxic element was disproportionately severe; a relation which probably had much influence on the production of the profound prostration, later profuse perspiration, and of the relatively severe nervous symptoms, manifest in insomnia, somewhat protracted though quiet, mild delirium, some peripheral neurolysis, and a variety of so-called "typhoid spine."

At starting, it is well to emphasise that much in typhoid fever is not direct pathogenic effect of its special bacillus. Sooner or later other pathogenic microbes are spurred into activity by the conditions in being, and may and do even secondarily affect the specific typhoid bowel lesions themselves. And a third important pathogenic group consists of various microbic and other poisonous substances produced in the alimentary canal in the course of typhoid fever, and all the more readily produced as part of the bowel is lamed by being the site of the specific lesions. The typhoid bacilli may be likened to swarming invaders who, besides the direct ravages they make, also loosen control in the invaded territory, and thus favour the setting free of local agents to spoil far and wide, and permit the gathering of a disaffected host in the very bowels of the land. Therefore not only many, but even the majority, of the symptoms and complicating morbid processes in an infectious disease, and especially in typhoid fever, may not be directly due to the latter's specific micro-organism, but to others, and even to some chemical

products of secondary digestive or metabolic disorder. That seer in pathology, Sir John Simon, long ago limited the specificity of disease to a vegetable or animal specificity. May we not now narrow disease-specificity still more, and say that it is merely the specificity of action of the micro-organism, or toxic, or other damaging factor, which starts the first one of a given series of morbid processes—a series in which other factors or other microbes may and do play important parts?

The Mental Wandering.

On the day of retirement to bed, and for a day or two after, there were, at first some headache, but wearing off; high temperature; light-headedness; considerable mental confusion and other evidences of wandering mind—all varying in degree at different times—and recollection of parts of those days is hazy. In this light-headedness and confusion the patient was unable to keep attention on any point, and therefore did not answer accurately and relevantly beyond a few moments. The mind was thus, as dove from ark, unable to find resting place, and like to sink from memory's ken in the trackless waste of delirium.

At this juncture, owing to a misunderstanding, it fell to the patient to transmit the instructions necessary to carry out suggestions made in his presence for treatment, for disinfection, and for remodelling the bedroom arrangements and conveniences. In this state of mental wandering—by dint of effort and of frequently regaining for a moment a sufficiency of the serial order, closer reciprocal ideal relations and intimate normal ideal associations of firmer and coherent thought,—he succeeded in imparting directions; but not without *many* breaks, excursions, and repetitions. Here was a striking, but perhaps not unusual scene; a wandering-minded patient communicating the instructions for his own treatment, the mental state remaining unrecognised by those present (non-medical). Fortunately the directions so given were appropriate. From this we may draw the practical reminder to be alive to the slightest indication of mental

wandering in invalids. Law may disregard the very little things; but, of them, clinical medicine must have a care.

And, of this state, another illustration occurred on the same day, when, in the course of conversation, "A" was questioned by a friend as to certain arrangements, and the latter was answered in a somewhat startling way, the bearing of his question having suffered delirious substitution, being replaced on the instant by a perverted interpretation that the question concerned arrangements for continuance of a marine structure deliriously imagined in course of erection, with mental toil and fatigue to "A," but as a project of that friend, with whom, therefore, and not with the deliriant, decision on the question ought to rest. And yet, by another turn of the wandering mind, the friend was understood as wishing the imaginary project to be carried out by "A's" instrumentality. Nevertheless, the reply made was so quick, decided, and apparently relevant; although, in truth, unfitting and surprising; that its delirious parentage was not even suspected at the time.

Yet the wavering Ego was even now partially and fluctuatingly confusing the self with others and with the surroundings. In confusion and seething turmoil, the Ego-group inclined to break up into constituent parts, to confound some of these with, or to attach them to, other persons; or to group the constituents afresh.

This is an early indication of the effects, on the systems of associational neurons, of the whole morbid operation of the disease; its toxæmia, fever, inanition, exhaustion by insomnia and over-activity. And, coincidently, degrees of motor helplessness, *e.g.*, degrees of inability for station and locomotion, marked a similar partial disablement of projection neurons, ministering to *action* in the life of external relation.

Continuing this subject of the disturbances and changes of consciousness of the self, of duple and multiple selves, and other affections of personality, it is of interest to note, thus early in the case, the peculiar confusion of self-consciousness; so that now and then the self, or some of the

self's possessions, interests, rights, responsibilities, needs or attributes, became confounded in the mind of the deliriant with that or those of another, or of others; *e.g.*, the patient confused himself in some respects with his interlocutor, as on the occasion just mentioned. But when he was thus partially changed into another, or into several others, the original self could usually be re-called, more or less, co-existently.

These affections of consciousness are important. Mostly, their start seemed to be in general mental confusion. This disturbed and broke through attention; and, the serial order of ideas, and their associative inter-relations, thus broken, the mutual, reciprocal, incitant or arrestive influence of latent ideas enhanced that effect. One result was a variety of strange dissolutions of the Ego-unity-group; at first, fleeting, indistinct, partial, or, if general, incomplete; but sometimes corrected momentarily and changed by any impression sufficiently intense to rally enfeebled attention; and then, when this slackened, mind again wandering and ready to turn back into the old way, or to stray along a new one.

Thus the Ego would be partly changed into another; namely, as regards some of its relations and attributes; or as regards its mental position and *rôle* generally.

Another stage observed at times was a partial division of the self into several new ones, the basis of each and all still remaining mentally rooted in the old.

Finally, at times attaining complete partition, the self became metamorphosed into two or more several persons. Withal, the recognition of these was variable; now distinct and vivid; again receding into distance and dimness; or, anon, disappearing.

And another phase which, not long after, came, was change of the self, general but incomplete; a reduction towards the mental status of childhood, a temporary child-ward mental retrogression.

At one time, the deliriant seemed to be mainly substituted by three persons, of different ages, all suffering like himself, from a bad attack of typhoid fever. At this point of time "A" had refused to take beef-tea, on purely medical

grounds, which at times were well within his remembrance during this time of light, quiet delirium from which he could, with ease, be aroused for the moment. And when expostulated with by the nurse, and the reason of refusal demanded; after some colloquy, his final reply was, "I'm sure it is not good for them." For *them*, be it noted. So that he was now, for the time being, assisting in the treatment of his changed and threefold self (really fourfold self); assisting, that is, by the candid expression of professional opinion; but from the view-point of the deliriant also exercising the far more effective part of forcibly modifying the diet of the imaginary three by refusing to take the actual beef-tea really offered to himself.

This may usefully remind us of the care to be exercised in noticing the least peculiarity in the expressions used by our patients. The change in the pronoun (*them* for *me*) was on that occasion the only indication of the mental wandering to the bystander. And yet it was a loophole through which apt and expert investigation would have revealed a wide expanse of delirious products largely occupying the whole range of consciousness at and about that time. The application of this is general, not individual; no medical attendant was present at the moment.

Let it be remembered that these conditions of changed consciousness of self were sometimes quickly formed and easily dissipated, occasionally underwent some transformation. They were apt to be recurrent, and in their reappearances either differing very considerably or fundamentally, or more or less similar, but never identical.

Never is the Ego twice precisely the same. In health, even, we hold and possess ourselves—each individual Ego—by no bond of absolute identity, but only by the ever-loosening ties of a relative similarity. From the adult self is ever exhaling something of the mental nature, part of the finer individuality, some of the latest gains of mental differentiation, a portion of the acquisitions and qualities of the self—difficultly renewed, or replaced by a satisfactory variant; and when decay and involution set in, renewable only in a

degree, or incompletely—a renewal ever minishing of acquirements ever deteriorating.

Lest the picture seem too gloomy, I hasten to add that age has its compensations, has its usefulness. Thus the moderating regulating influence of age in council has often well served the State; so also has its strategy in the field, as witness a Moltke or a Roberts.

Affections of Nose, Mouth, Teeth, General Sensibility, in relation to Mental Wandering.

Somewhat early in the case, and continuing for a time, perverted sensibility of various parts and organs existed. Paræsthesiæ and irritation of nasal mucosa (fifth nerve) were potent sources of delirious notions. The mouth was the seat of many paræsthesiæ; the teeth felt loose and altered as if to assume a difference, not so much of locality as of semi-independence, as if they were loosened from their moorings and half free-floating in air, and yet taken to be in the mouth and mainly in their usual relations to the adjacent parts; speciously, also, communicating a feeling as if they were soft bodies, possessing sensibility. A feeling of somewhat general pulpiness of the body lasted for some days.

The perverted sensibility of the mouth and gums, the paræsthetic somewhat general pulpiness of body, and the suggestions flowing from milk diet, then in force, were eventually connected with strange delirious episodes of childish type; also connected with various false perceptions, as, *e.g.*, of coloured glass, of pressure on teeth; constituting a reversion to childhood—associations of second teething, toothache, coloured glass ornaments, cows yielding milk, and so on. Thus the reduction in this phase was to childish associations, to children's thoughts and ways of viewing these experiences.

Early marine structure-building.—Reference has been made to the structures raised on the sea or harbour of the delirium and shore adjoining. Partaking of the phantasmagorial nature of their parentage, their excitation was of a

more or less shifting variable kind; and at first they were skeletal constructions of timber, a ready-made material from the impressions of youth, when the raising of such structures, and the completed open structures themselves, were often seen, and therefore were much stored away in the early strata of mind-life. And now and then, in imagination, such structures were raised, not without seeming effort on the part of the mind-wanderer.

Other phantasms.—Various illusory and hallucinatory deceptions thoroughly imposed themselves on the mind for varying spaces of time thereafter. One experience was that "A" seemed to be moving on the winding paths of unfamiliar landscape gardening. A number of hillocks or mounds were beset with pathways winding in all directions on the slopes, and ascending or descending, mounting the hillocks, or descending from their summit to the small depressions between them; and ever winding and crossing on the acclivities and declivities. And between these pathways, always winding on the steep slopes, were numerous small trees or large shrubs with unusual foliage and fruit, in fact, all of a soft pulpy appearance, reminding of waxen artifice. The colours of foliage and of the sparse fruit were subdued, many dim hues more or less brown; some gray, and some dim subdued blues and green.

At this time and thereabouts the head felt light, dizzy or vertiginoid, and insomnia was apparently complete. No sleep came to refresh. This gave anxiety to those in attendance, and often the patient, when clearer, attempted to compose himself to sleep, understanding the desirability of that restorative urged on him, and also anxious to sleep in order to avoid narcotic drugs, none of which, in fact, were taken.

This phantasmal phase seems exceedingly difficult to analyse. But the deliriant's movement on winding pathways which beset the bold hillock-and-hollow configuration of the ground, this locomotion on undulating surface, and the inseparability of the two (namely, up and down movement and ground form) at the time, betoken the swaying vertiginoid state of consciousness. And certain co-incident

conditions indicate a clue to further insight. For at this time there was a more or less general condition, varying in degree locally, namely, a feeling as of pulpiness of parts of the organism—an easy delirious assimilation of ideas, in the existing state of partial Ego-dissolution, endowing that false environmental appearance with something analogous to that felt attribute of the self's body pulpiness. And another co-existent condition was that of insomnia, here to be taken in relation with former experience of impairing and disordering effect of railway sleepless night travel on "A's" colour sense.

Without claiming to explain, fully, the colouration of this imagery, it certainly is interesting to note that, coincidently with the toxic and starving results of the fever and of the insomnia, modification of the colouring of the apparition was equivalent to that effected in actual sensation by reduction of the light-intensity of stimuli of colour sensation in normal condition—by failing response to the stimuli in abnormal—namely, a reduction of colour sensations more or less, on this occasion less, toward sensation of black. This would account for all but the green; which, however, may easily have been an inferential addition to the phantasm, or to the memorial image of its foliage; inferential addition analogous to that we all make, and a thousand times, in perception and memory of objects and occurrences.

We have referred to the phantasmal marine construction, also to the hillock-and-hollow episode. At a later and more advanced phase the swaying, yet mainly fluctuant and vertiginoid, state of consciousness was allegorised by the disturbed associative processes into delirious conceptions of being out on wild waters in a vessel tossed about by the waves in a regular and rhythmically recurring up and down movement, rising and falling in large undulations.

Thus it was that, when greeted on one occasion, "A" gave an account of having been out in a boat much tossed about in the seas, so that he had expected it to capsize.

This rhythmic recurrence involved (1) sensation and ideas of a particular kind of total motion in space of a comparatively passive body (although, no doubt, some fine

appropriate balancing adjusting muscular tensions occurred therewith); and also (2) ideas of spatial relations; involved, therefore, in a certain measure, what one has supposed to be a function of a certain large cerebro-cortical realm.

This regular and rhythmical affection of consciousness seems to demand a factor regularly and rhythmically recurrent at that time, and a correspondently rhythmical fluctuation in nutrition and function of the particular nervous elements concerned. And we turn to the brain circulation, naturally. So far as one could gather of the rate of apparent fall and rise, alternately, in this long billowy swell, from crest of surge to trough of sea and back again to crest, it seemed doubtful which may have been concerned of the two possible, namely, vaso-motor and respiratory, out of the three normal regular fluctuants of the brain circulation—producing waves in the blood within the great intracranial conduits. The conditions in this case were unsuitable for deciding the question, but some future example may yield the requisite relations, and that is why the question is raised here. For (if I may be pardoned a transient lapse into the hypothetical) two hypotheses at once present themselves:—(1) that toxæmia may affect vaso-motor variations of intracranial vessels so that they become morbidly mutable. Then the above rhythmic vaso-motor undulations would represent a variety of Traube-Hering waves in excess. On the other hand (2) rhythmical disorders of respiration are not difficult to produce either pathologically or experimentally, and may exaggerate respiratory undulations in brain supply.

Neither the question of the existence or non-existence of vaso-motor nerves for brain, *nor* that of abdominal vaso-motor influence on brain circulation, can be taken here.

Temporary bronchitis itself, and the effects of the inhalations used for it, were, or seemed to be, irritating; and these effects became duly translated and wrought up into various delirious notions, as, for example, of being anæsthetised or suffocated—notions which, in their turn, led to vigorous expostulation and denunciation by the patient.

The abdominal tympanites and pain of the general disease, *i.e.*, of the enteric fever, had their share in shaping the mental wandering, receiving interpretations varying from time to time, rapidly or instantaneously conjuring up conditions of suffering, of pain, of fleeting, evasive, dimly apprehended danger or attack.

Real and imagined sounds heard—such as voices or laughter—broke in strangely and unexpectedly, at times, upon the course of an ambling delirium.

But, necessarily, the *visual* impressions were a centre of much perversion. Especially when, in the flickering light of the fire in the large open fire-place opposite to the patient lying a-bed, the small china statuettes on the mantel-piece seemed to undergo a thousand transformations; retaining no set form, but as if moulded and twisted every moment by an inward plastic power, making them ever mobile, and yet always in definite relations to the transformation and movements of each other, like contortionists in lively rhythmic and incessant change, each movement of which is in unison with, and complemental to, appropriate movements in the fellow-piece of each pair. Attention of enfeebled mind, at times, was as if fascinated and held fixed by the sinuous writhings and contortions of this ceaseless, wearisome dance of two, until the eyes were kept resolutely shut or the head was turned away.

And the lights and shadows cast by the open coal fire, as it rose and fell, or threw out fiery tongues hither and thither, were in other ways a potent source of delirious conception. From time to time they brought out ideas, such as that the scene was in a dome-like edifice, in which fires mysteriously burned, and where gaily-dressed persons fluttered about; at another time, that in it was a small yacht, and in that vessel a cannon covered with flags, and the deliriant and others were taking part in a wily and successful ruse to conceal the cannon, and thus outwit the foe; or, in the dome-like space were lightly-clad figures, somewhat transformed gloriously.

Modification of Delirium by Sleep-dream, i.e., Fusion and Transmutation into Delirious Dreaming, or Dreamy Delirium.

Passing to another and later stage, we must now refer to the influence of sleep and dreaming on the clinical aspect.

After the somewhat protracted insomnia; the return of sleep, at first in a light, fitful, broken form, modified the mental state. For the wandering of mind, delirious fancies, and comparatively fleeting phantasmal appearances of the earlier period—with its insomnia, its confusion, its dizzy or vertigo-like state—became both modified and exaggerated in the dreamy states of this partial and defectively-refreshing sleep. Under *this* sleep-dream influence, the illusory and hallucinatory phenomena assumed a greater sensual vivacity; often an exaggerated, or even, sometimes, absurd expression; and there came into play more intense emotion, and wilder fluctuations in its range between the possible extremes.

I think we do not assign to dreaming states in disease all the importance they really possess, whether as symptoms, prodromic, or of the established malady; whether as modifying, enhancing, or precipitating scenes and acts in the morbid drama of delirium, or of insanity.

The phenomena of dreaming on the one hand; the illusory and hallucinatory elements of delirium on the other; have some close resemblances when viewed apart. And in the sleeping state of deliriants, such as it is, the delirium and dreams seem to fuse into one; nay, more, to be metamorphosed or even transmuted into one. Then we may speak of delirious dreaming (or of dreamy delirium).

In some dreams of disease the psychological condition is much like that of the illuded and hallucinated deliriant in waking state. Hence a resemblance to a dream-scene in the awake state.

Moreover, dreams are constituent elements in a graded scale we may construct, from the one extreme of the normal sane waking condition, to the other extreme consisting of the set hallucinations and delusions of technical insanity.

Normal sane, waking state.
Dreams in health, or fair health.
Hypnotic and somnambulic sleep, with illusions and hallucinations.
Dreams in, so-called, ordinary disease
Quiet delirium of ordinary disease } may unite.
Active excited delirium of ordinary disease.
Initiative delirium of technical insanity.
Episodial delirium of technical insanity.
Set hallucinations and delusions of technical insanity.

As illustrating the sometimes grotesquely exaggerating nfluence of delirious dreaming, it may be mentioned that on more than one occasion, when he broke out in real and profuse perspiration, the patient seemed to himself to be like to drown in, or to be carried away by the tide of, his own perspiration.

These gross exaggerations of the amount of the sweats—profuse as these were in reality—were carried over into the time of full awakening, correction being attained after the lapse of some appreciable time; another example of the points of touch between certain dreams and mental disorders, and reminder of the influence of the one upon the other.

Comparatively early, and in mid-period, had certain features presented; a recurring wearisome monotony of slow hours of delirious consciousness, a more or less painful, weary, cyclic recurrence of events, as if in a procession of the hours; each period of which had its type of morbid idea, its favourite and predominant kind, and even sometimes individualised set of ideal associations.

But chiefly was there a mental movement in an oblong quadrangular course or circuit, and not in a circle proper. With painful eagerness was it sought to carry out some act of recollection, or some imaginary work or project, or to supervise, in this circuit, whether concerning the toilsome progression of vehicle, the process of flagging the highway, the bridging and telegraph construction on the line of road; these changing from time to time, and often only vaguely remembered in particulars afterwards. The deliriant's actual

conveyance, and really under his own directions, to a close-stool beside the bed seemed to him to be a triumph of engineering skill directed by him, and achieved with the aid of great personal effort and strain on his part. And here again the quadrangular element came in, apparently suggested by the square-set arms and back of the night-chair.

Later on was a frequently repeated experience. A square-set room on wheels performed a regularly recurring journey on an oblong quadrangular course. Steadily, but heavily and awkwardly, and with much shaking, concussion and vibration, did this awkward lumbering wain make its weary monotonous quadrangular round. And the wain and its processions were tacitly accepted as constituting a symbol. Though scarcely put in words, there seemed here to be a simile or an abstract summary of the experiences of that time; indeed, more than such simile, being in fact most intimately associated with and representative of the self, and a part of the existence.

The shaking of the imaginary wain was the exaggerated and altered interpretation of the real vibrations and clatter of passing railway trains, of waggons in the adjoining roadway, and of the actual demolition of a brick building; while the shape of the course of the weary processions was the same as, and apparently suggested by, the similar shape of the large bed in which the patient was lying for weeks, and of the room in which it stood, the longer and shorter measurements of the two coinciding in direction.

Thus these mental phenomena sprang from impressions issuing from the neighbourhood, fused with stereotype impress of the immediate surrounding.

Another experience concerned a structure with lines of timber, iron, and wire-rope strings, on inclined planes, rising obliquely—an instrument, vast, far-spread, and taken as analogous to a mechanism symbolic of myriads of human lives settled thereon; suggesting a prodigious, bewilderingly complex violin of human life and kind, each of the million cable-strings of which had its intense life-meaning, to touch any part of one of which indicated some individual's position

and *rôle* in the social organism and in the moral and intellectual spheres, and expressed his life abstractly, in a critical summary.

Entangled on these obliquely-set cables or slanting lines of life, only able to modify the position without wholly controlling it, without the ability to alter it essentially—though by constant effort and strain some modification and improvement could seemingly be effected; and, conversely, by laxity or carelessness and effortless acquiescence, the position could worsen and tended to do so—one had here, apparently, a mental equivalent in the summarising of the deliriant's existing state of comparative physical prostration and helplessness, whilst he was lying a-bed with half-raised head and shoulders; and these conditions projecting in relief on the groundwork of a summarised sense of existence, both physical and mental.

Emotional States and Disorders in the Case.

In the production of these, also, I attribute an enhancing influence to dream consciousness (in dreamy delirium).

Comparatively calm *neutral states of emotion.* Not at all times is the mental wanderer a prey to emotion. The degrees of feeling, whether expansive or depressed, wane from time to time, and then approach or enter into what we may liken to a haven of rest shut off from the wildness of emotional storm. At these times the patient apparently becomes a more or less impassive spectator of the visions.

Expansive phases of emotion: states of exaltation.—From time to time throughout much of the wandering, the mental associations seemed, speciously, of morbid liveliness; the strict logical boundaries were easily overleaped, and much of the day-dreams, and points of satisfaction in life-history of ancestry or of the self, now and again rose to the surface.

Even amidst the weary monotony of afore-mentioned recurrent experiences were schemes and projects repeatedly conceived; and every now and then came the feeling of being able to promptly accomplish various designs; whereas,

in reality, the patient was weak and helpless, incapable of getting up, or of walking or of standing for an instant. Herein is a condition to consider in relation to the morbid feeling of capability and power in mania, and often in general paralysis.

In the wide-awake consciousness of the established positive emotional phase, there was emotional and ideational expansiveness, with the degrees of quiet optimism, serene exaltation, uncurbed merriment; and with its rapid, loose, incoherent ideation. This expansiveness was influenced by the oncoming of light defective sleep, so as to be modified and transformed or transmuted into delirious dreaming; in which, while the preceding rapidity and looseness of ideation became shifting kaleidoscopic change, the emotional expansiveness of the awake state became the grandiose of the dreamy delirium, and the patient as one drunken with joy and power. In this way expansiveness attained its further degree of grandiose inflation.

Presently we come to description of this inflated period of the grandiose. But before that phase, the patient dwelt in slow transformation scenes, the scale of which was the measure of greatness. At first were glorious marble palaces of the most exquisite proportions and beauty; noble and stately temples; these triumphs of art held in the setting of lovely scenery, with hill and vale, stream and lakelet, mountain and forest, torrent and waterfall; with the far distances magnificent.

Yet, later on, there was a falling off from the serenity of this exaltation; marking, no doubt, a variation in the kind or degree of the toxæmia, or in the resistance offered to it by the nervous substance, as well as a lowering of vitality. This declension from the state of calm serene exaltation befell from time to time, and was duly weaved into the allegory of the delirium. So it was that ruin partially lighted on these stately piles with their enchanting surroundings. Tumult and quarrel and the destruction attending civil warfare, brought change of scene; some of the glorious palaces fell into ruin, others showed traces of the stress of warfare, and around them came the signs of neglect and

destruction; as, for example, the wreck of what had formerly been charming wire work marking divisions of the scene, but now cast down, and lying torn and outspread in a disorder which impeded the progress, snared the feet, and entangled the movements of the passer by.

There was much difference between the scenes attending serene exaltation and certain other phenomena—speciously and superficially similar—immediately following a later part of an imaginary experience of making cold, snowy journeys in wearisome circuits (and not described in this address). For although in the latter the scenes contained grand architectural erections, these seemed to have a significance and use mainly monumental or symbolic; they were the emblems of certain influences and forces, and bound up with the personality and life-relations of certain persons more or less notable; and the difficulty in accomplishing some designs in reference to these monuments marked a crisis in existence, and symbolised failure or success. Moreover, these scenes and their columnar embellishment were far more set, fixed and stable than those of the exaltation a few moments ago outlined. Whereas these last, indeed, were usually in some degree of change, had about them more of the spectacular than of the symbolic, marked an easier course of mental motion, a livelier fancy, which at times was dramatic in action, swift in change, fleeting in duration. Contrast with this the sober, earnest, striving effort and symbolism of the other group of scenes with imposing architectural elements.

Some time after the start of the more fully expressed expansive phase of feeling it passed into the stage of inflation already mentioned, this being somewhat variable in degree. Harmonising with the conceptions, were pervading feelings of joy, efficiency, of ease to do surprising feats, of superabundant power, actual and potential; a delirious, inflated, lightheadedness, as if the blood had been turned into glorified champagne; gay scenes of irresponsible merriment, laughter, music, merry pranks; and the swelled sense of greatness

and easeful power of body and mind; in flagrant contrast with the smitten paresified reality. And then, through association of ideas, like a flash, the revelation that all this was morbid and a mockery. And then, in the burst of an maginary chorus of laughter, the thought of "A" that he had become general paralytic, and the comment aloud recognising and naming the condition as "General Paralysis." A blundering diagnosis, but the man was then distempered in mind, and much must be forgiven him. Indeed, the mistake was no worse than some I am aware of, and without that excuse. General paralysis, with its grim, imminent fatality fully in view, and yet no shock suffered from the knowledge, no concern even felt; it scarce gave him pause from the mental rollicking and rioting in chimerical happiness. On the contrary, there was yielding to a bubbling-over merriment; this inflated viniform state reminding so strongly both of general paralytic exaltation and of alcoholic, that it may be, in part, aptly represented by a thought which was then actually in the patient's mind and phrased in the quasi-alcoholic self-exhortation, "Never mind, it's all right, *must* die *some* time."

Later came a phase in which "A" imagined himself to be whirling down the mountain slopes; or to be swiftly diving—literally diving—down through huge trees, from top to bottom, rending and peeling off thick layers of the ringed wood, and hurling them aside, as if by lightning-stroke. Yet the *absence* therewith of any delusion of being changed into a thunderbolt was grossly illogical!

These various expansive symptoms have a complex origin. They mark an unwonted and morbid incitation of, at least, large cortical areas, the substrata of ideational, kinetic and sensorial activity.

Surely with the toxæmia we have a chemical effect of transcendent fineness, working on delicate susceptibility of nervous elements, which in some examples with greater intensity of intoxication may attain to grave lesion, either by summation of effects, or through conditions promoting

concentration of these locally; whereas in other examples the influence remains diffuse, light, fleeting, and the concomitant neuronic changes more or less easily recover. Sufficient to produce neuronic change is any one, alone, of several factors present: namely, toxæmia; febrile temperature; brain inanition; brain exhaustion of insomnia; to say nothing, here, of a secondary self-poisoning which may spring from each and every one of these; nor to mention the hypothetical brain anæmia.

Thus the agents are, the direct inciting, disturbing and altering effects of these several factors on function and structure of nervous elements. The accompanying disturbance of brain-circulation, impressions from the body and from beyond it, and the modification and enhancement by sleep-dream, all play their part.

On the other hand, it is in the emotionally depressed phase, yet to be described, that we find the psychical concomitants of the exhaustion and profound vital lowering of the organism and of the same poisoned neurons; a phase, as regards its extremely brief but severe manifestation, occurring later in time, although earlier, milder and transitory indications of morbid negative emotional tone had often been interposed.

The Transitory Phase of Emotional Depression.

Eventually appeared an exceedingly brief time of mental misery and despair. Nor was the advent of this entirely unprepared: the anxious straining efforts, the mental and physical malaise, and the gloomy promise existent in some of the preceding stages of the malady, had intermittently overcast the mental firmament somewhat, and prepared the way for the temporary submersion of consciousness in gloom and blackness. This, too, was the very time when the patient was emaciate, helpless, sunken in prostration, shadowed by the wings of death: nutrition at a standstill, vitality at its lowest ebb, so that for days together no gain towards recovery was made.

In this process of transient submersion of consciousness in gloomy blackness, quickly the deeps of woe were reached, than which it seemed no lower still *could* be. Then the Ego faced what was, for it, vaguely understood as a final state, a sempiternal woe and misery.

For our mental wanderer seemed to be now lying prostrated, in physical exhaustion and mental misery, amid dim and murky surroundings; and, gazing upwards, descried slowly revolving wheel-like concentric sets of metallic rings beset with numerals and other signs and figures. And it came to him as if each of these symbolised a person; and one among them was understood as being his symbol, and two others as those of two among the dearest to him in his life; while the rest were understood to represent others.

In this dim and dismal region hope was effaced, escape was impossible, and so was death; out of reach the heaven of annihilation. This misery, intense, and in prospect eternal, was taken as essential misery, and not as punishment.

Exceedingly brief, indeed transient, as was this phase of the depression, and lasting a small fraction of a day: what it lacked in duration it more than made up by intensity. It seemed as if the man was, for ever to come, fast in the pitch of the blackest pool in some grotesque mediæval inferno. And ever and above the wheeling symbols kept circling in their eternal motions.

Neither has ear heard, nor tongue uttered, the wretchedness of this state of despair.

Happily, this transient, gloomy, dismal scene may be taken as metaphorically representing the darkest hour before the dawn of a new time of continuous mental light and life.

Nevertheless, for some weeks thereafter, the continuance of helplessness, prostration, colliquative perspirations, finally with pain, made the nocturnal life, from midnight to seven in the morning, most wearisome and uneasy. But herein the predominant physical condition merely translated itself in a sombre shading of the mind—now perfectly clear intel-

lectually, although easily fatigued, and no wonder! Indeed, like any little physical exertion, so also slight mental work, and particularly some really trivial, but now large-looming, annoyance, crossing or thwarting, increased the pulse rate, raised the temperature, resolved in perspiration; another proof, to add to the thousand already possessed, of how intimately, intricately, inextricably intermingled and interwoven are the mental and physical in man.

Contrasted with recollections of one's reading, the distinctive features of this address chiefly concern :—

(1) Partial or complete transitory division of self-consciousness, and even duple and multiple personality, in mild, quiet delirium, or mental wandering.

(2) Attempt to analyse the phantasmal experiences and apparitions.

(3) The transforming or metamorphosing or transmuting effect of sleep-dream on illusory delirious states; or of these into one.

(4) The lively, vivid, emotional play and show in mental wandering.

Minor points might be added to these, but it is better not to distract attention from the chief ones.

Subjects Omitted.

Limits of space and of time have compelled brevity and incompleteness in dealing with some, and total omission of many, of the subjects raised by this case. The omissions are chiefly as follows :—

(*a*) Further considerations on changes in consciousness of the self.

(*b*) Discussion of peripheral neurolysis in this case, also in reference to so-called polyneuritic psychosis, and to evil effect of cold, and beneficial of warmth, in toxæmia.

(*c*) The bladder symptoms, dysury, &c., and their accompanying mental symptoms.

(*d*) The constipation and dyschezia in relation to sense of effort, &c.

(*e*) So-called "typhoid spine" in the case. Reasons for rejecting the name.

(*f*) Dream-states in relation to mental disorders.

(*g*) Prominence of visual elements in the symptoms.

(*h*) The great variety of the symptoms.

(*i*) Symbolism in delirious phantasy.

(*j*) Mental wandering, as presenting comprehensive summary-sketches, foreshadowing many forms of mental disease.

In conclusion.—When we ponder on the basis of mind, it must, for us, be a lesson in humility; so imperfect the basis that mental error is more easy to understand than mental accuracy. How light our hold upon what we possess mentally; how easily that tenure relaxed or lost! A trifling disorder of health affects mind more or less, every grave disease affects it much; even in ordinary conditions the measure of mind *available at a given time* varies exceedingly; is bettered or worsened or modified by atmospheric and barometric conditions, sunshine or cloud, the state of a function or viscus, the cyclic change of day and night, quality and amount of food, kind and length of sleep, mental and moral incidents, and a thousand external circumstances. A tiny particle of many a substance can clog or disorder the working of mind, or obliterate it temporarily, and certain lesions permanently. A slight change in the chemical constituents of the blood can raise us to the height of bliss, plunge us into the depth of despair, or, as in our case to-night, with light delirious touch strike many a note in the scale of morbid mind.

Appendix.

At the beginning of this address the general symptoms of the enteric fever were brought down to the fifth day. A brief summary of the subsequent course of the disease is added here.

Besides the delirium, and for a time sleeplessness, the prominent symptoms, thenceforward, were: rose rash, diarrhœa, tympanites, abdominal pain, dysuria, emaciation, as-

thenia, profuse perspirations, some peripheral neurolysis in limbs, bronchitis, congestion of lungs, slight contracture of lower limbs, &c.

By about the thirty-fourth to forty-first day emaciation had become excessive, prostration extreme, constipation had replaced diarrhœa. Peripheral neurolysis had left some muscular wasting, motor weakness, and slight hyperæsthesia of parts of hands and feet; no improvement in general nutrition was taking place. About the forty-first to forty-third day the wise and successful restrictions in diet could be relaxed and set in train for abolition. Improvement slowly set in. About a week afterwards a variety of "typhoid spine" began to come on gradually, at first with lumbago-like pain, and, subsequently, arthralgic pain.

For a time perspiration at night had been, and continued to be, profuse, dysuria troublesome, the evenings drowsy, and the night-morning (midnight to 7 a.m.) restless and depressed; the legs still contractured, though improved by massage; and the pain-aches, and pain-shocks of lightning type, and susceptibility to chill, were still pronounced.

Throughout the two months following the forty-third day (a month-end), constipation and racking strain at stool continued; inability to walk or stand persisted, and, during the latter three-fourths of this space of time, pain on movement precluded any attempt to do so. The body continued to regain weight. The relics of peripheral neurolysis, possibly with a degree of cornual implication, consisted of atrophy and fibrillary tremors of certain muscles, some local defect of power, numbness and paresis, easily made worse by cold. At the end of that period (hundred and fourth day), the patient was taken to the seaside. A fortnight later the first step was walked. Afterwards bath-chair exercise was taken, in short turns only, inasmuch as, in the sitting position, discomfort, aching and pain about pelvis and buttocks resulted from the pressure of weight of upper part of body, even when the softest feather pillows formed the seat. Walking power gradually became re-established in the course of the following month (hundred and thirty-fourth to hundred and sixty-fifth day).

Temperature.—Unfortunately, the temperature records of the first few days are mislaid or lost; the highest point recorded during that time was 104° Fahrenheit. The charts in hand begin on the date six days after the first slight deviation from usual feeling of health. They end on the eighty-seventh day. The temperature was usually recorded six times in the twenty-four hours.

On and after the seventh to sixty-third day, the charts show a general tendency to rise in the afternoon or evening, or both; to decline considerably in the latter part of the night, *i.e.*, 2 a.m. to 6 or 7 a.m.

But there are some erratic deviations from this rule, and occasionally the temperature was moderately high (or even highest for that particular twenty-four hours) at about 11 a.m.

Seventh to twelfth days (inclusive) six days; no temperature below 100°; the range practically 100° to 102°; highest 102·5°—thirteenth to twentieth days, eight days; on a somewhat lower level again (about 1° lower), evening rises and morning falls continuing.

Twentieth to twenty-fifth days, temperature on average just below normal, and only once as high as 100°. Then on twenty-sixth or twenty-seventh days erratic rise to 101·6°, and remaining high for some hours. On twenty-eighth, twenty-ninth and thirtieth days, the temperature again down (as twentieth to twenty-fifth), then a rise to 100·6°. Then for three days the temperature constantly between 99° and 100°, day and night. Then the same (with occasional erratic rise) to the fortieth day. Then for several days subnormal mostly, and not much fluctuation.

After this several marked rises, 101°, 101·6°. Later on, subnormal for two days.

Later came tendency to late morning, midday, and early afternoon rise; but sinking in late afternoon and evening, lower and lower, and so in early succeeding morning hours, until the diurnal rise recurred (say) by 10 or 11 a.m., midday, or 1, 2, or 3 p.m., and then soon falling again.

From sixty-second to seventy-fourth days, average

temperature slightly above normal, sometimes being 99° to 100° in evening.

From seventy-fourth to eighty-seventh days, temperature normal, or only very slightly above normal.

Diarrhœa: from about seventh to twenty-fourth day, and subsequently returning for a few days.

Constipation, from about thirty-third to thirty-ninth day (inclusive): enemata. Then a few days looseness of bowels, followed by two months of more or less severe constipation, requiring frequent enemata and laxatives *p.r.n.* In the month following the bowel habit was in process of becoming normal.

Urine: stated to be free from albumen and sugar at several examinations.

Diet during fever: mainly milk, eggs, and some beef-tea.

EXPERIMENTAL LESION OF THE CORPORA QUADRIGEMINA IN MONKEYS.

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In the course of our investigations upon the symptoms and degenerations following experimental lesion of the several portions of the mid and hind brain, elsewhere described (1), partial lesions of the quadrigeminal bodies and of the subjacent tegmentum were produced. It was therefore considered advisable, both from the importance of the anatomical connection of these bodies, as well as from the variable and uncertain results obtained by previous investigators, to make a further series of experiments with a view to their complete, and as far as possible limited, ablation. In the present communication the symptoms of our experimental lesions will alone be described, leaving for a future communication an account of the degenerations so induced.

The functions of the corpora quadrigemina have been the subject of many researches, but considerable uncertainty still exists as to their true physiological significance. There seems to be an inverse relation between the development and functional importance of the quadrigeminal bodies—optic lobes of fishes, amphibia and birds—and the cerebral hemispheres; and in the second place the anterior and posterior pairs have respectively different anatomical connections with the other parts of the central nervous system. Hence the experimental data may be expected to vary according as the observations are made upon lower vertebrates such as fishes,

frogs and birds, or on the higher mammals such as monkeys, and as to whether the lesions affect the anterior or posterior pair or both.

From a review of the earlier literature upon the functions of the optic lobes or corpora quadrigemina, as observed by the method of destructive lesions, the results may be summarised under the following heads.

(1) *Vision.*—Various observers, among whom may be mentioned Longet (2), Flourens (3), Schiff, McKendrick (5), Ferrier (6) and Bechterew (7), found a constant relation existing between the anterior lobes and the sense of sight. Destruction of the optic lobes gave rise to visual defect, but the extent of the loss of vision was dependant upon the complete or partial decussation of the optic tracts. In those animals whose vision was monocular with complete decussation at the chiasma, removal of an optic lobe occasioned blindness in the opposite eye; while in those with binocular vision, there resulted hemianopsia to the right or left according as the left or right anterior bodies were destroyed. The experimental observations of Bechterew upon dogs are the most complete in this connection. For example, destruction of one anterior body gave rise to homonymous hemianopsia to the opposite side, and a like result followed unilateral destruction of both bodies. The pupillary reaction was only affected if the lesion extended deep into the Sylvian gray matter. A complete lesion of both anterior bodies produced complete blindness. This last observation is in harmony with one by Ferrier, who found complete blindness after destruction of both anterior quadrigeminal bodies in a monkey.

The more recent experimental observations of Fano (7A), upon testudo, and of Stefani (4) upon birds, showed that although there was some impairment of vision, as a result of destruction of the optic lobes, it was clear that vision was not entirely abolished.

The results of these experiments therefore are in favour of the view that destruction of the anterior pair, whether in the lower vertebrata, such as fishes, frogs or birds, or amongst the mammalia, such as dogs or monkeys, is followed

by impairment or abolition of the sense of sight. The observations of Nothnagel (8), on the other hand, upon the effects of disease of this region in man failed to prove any interference with vision which could be ascribed to lesion of the quadrigeminal bodies as such. In a case of bullet wound of these bodies upon the right side, recorded by Eisenlohr (9), there was no affection of vision immediately after the receipt of the injury. Although the relation of the optic lobes to vision may be clear in the lower forms of vertebrate life, it is by no means so certain between the corpora quadrigemina and the sense of sight in the higher mammalia. The facts which we bring forward in this paper make it clear that ablation of the quadrigeminal bodies as such in monkeys is not followed by any impairment of vision.

(2) *Reflex action of the pupils.* — Some observers—Flourens, Longet, and others—found that the destruction of the quadrigeminal bodies caused, in addition to loss of vision, dilatation of the pupil and loss of the pupillary light reflex—an observation which indicated a centre co-ordinating retinal impressions and irido-motor action. On the other hand, Knoll (10), experimenting on rabbits, and Bechterew on dogs, did not confirm the pupillary immobility after destruction of these bodies. It was only observed by the former when the optic tract was itself injured, and by the latter if the lesion was sufficiently extensive so as to involve the gray matter around the Sylvian aqueduct.

(3) *Associated movement of the eyeballs.*—The phenomena observed upon stimulating the quadrigeminal bodies —Ferrier, Adamuk (11), Bechterew—show that, in addition to tonic spasm of the body and limbs, ocular movements of a definite character have been noticed, such as dilatation of the pupils, opening of the eyes and elevation of the eyebrows, and conjugate deviation of the eyes to one or other side. These phenomena have been ascribed by some to diffusion current, as some of the results may also be obtained by irritation of the subjacent tegmentum after the quadrigeminal bodies have been removed. On the other hand, it has been claimed that the results of stimulation are due to irritation of the quadrigeminal bodies, as such; for the

effects of irritating the anterior pair differ essentially from those consequent on irritation of the posterior pair.

This view would seem to receive confirmation from the recent stimulation experiments of Prus (18). From his experiments, which were made upon dogs without narcosis, this observer concluded that in the anterior and posterior bodies respectively are centres for influencing visual and auditory impressions, that in the anterior pair there is a centre for tonic, and in the posterior pair a centre for clonic convulsions, and that through these bodies there pass reflex paths for respiration and vasomotor action.

(4) *Maintenance of equilibrium.* — Serres in 1827, first pointed out that destruction of the corpora quadrigemina in various animals gave rise to marked instability and disorders of equilibration. These observations were confirmed by numerous investigators—Cayrade (13), Goltz (14), Kohts (15), McKendrick, and Ferrier. Thus fishes, although able to swim, could not maintain the normal attitude: frogs rolled over and over and were unable to balance themselves; pigeons, when thrown into the air, oscillated and fell down, and rabbits showed marked instability during locomotion. On the other hand Bechterew, experimenting chiefly on dogs, failed to find any disturbance of gait or locomotion after destruction of the quadrigeminal region. When these defects were present, the subjacent structures had been injured, and more especially the superior cerebellar peduncle.

A reeling gait has been described as characteristic of disease, especially tumour, of the quadrigeminal region in man. In appearance it resembles that seen in cerebellar disease rather than that of locomotor ataxy. If the disease is progressive, locomotion and the maintenance of equilibrium eventually become impossible. These symptoms have been attributed to implication of adjacent structures, rather than to interference with the functions of the quadrigeminal bodies as such. In the light of our experiments here recorded, where no ataxy or disturbance of equilibrium was observed—provided the lesion did not extend beyond the limits of the ganglionic structures—this would seem to be the true explanation.

(5) *Hearing.*—Destruction of the posterior pair was found by Bechterew, experimenting on white rats and dogs, to be followed by defect or abolition of the sense of hearing, according as the lesion was partial or complete. Only when the lesion was quite superficial did hearing seem to be unimpaired. The close association which is known to exist anatomically between the posterior pair and the trapezoid body, by means of the lateral fillet, would appear to point to an important relation between these structures and the sense of hearing. But even under the most favourable conditions it is difficult to estimate with any certainty the degree of deafness in animals; and in disease of this region in man, in which the state of hearing was specially investigated, the results are very uncertain. In the only one of our experiments (No. 2), in which the facts seemed to warrant the existence of deafness, the lesion was so extensive that not only the quadrigeminal bodies, but also the tegmentum and the central auditory tract, were destroyed. In experimental cases, therefore, in which hearing is said to have been abolished, unless a subsequent microscopic examination has been made, the direct dependance of deafness upon the quadrigeminal lesion may be open to question.

(6) *The voice and emotions.*—Some of the earlier investigators, more particularly Goltz, were of opinion that the quadrigeminal bodies are a centre for emotional expression, but the more careful observations of later experimenters have not corroborated this view. Bechterew states that ablation of the anterior quadrigeminal bodies does not produce any affection of the emotions, but that, if the posterior pair are destroyed, weakness of the voice may ensue, and that it may be completely lost if the lesion is bilateral and extensive. Ferrier, on the other hand, produced extensive lesions of these bodies in rabbits without causing cessation of the characteristic cries evoked by sensory stimulation.

Onodi (16) also has separated the quadrigeminal region from the higher part of the brain without interfering with the approximation of the vocal cords; while Sherrington (12)

found vocalisation abolished after transection immediately behind the posterior bodies in monkeys.

(7) *Bodily temperature.*—Some experiments by Ott (17) upon puncture of the quadrigeminal bodies in rabbits showed a rise of from 1° to 2° F., which lasted for three or four hours. According to this observer, these effects were due to disturbance of a thermolytic centre in this locality. Irritation of the quadrigeminal region has also been found to increase the blood pressure, as well as to modify the respiratory rhythm.

Method of Operation.

Young monkeys—*Macacus sinicus* and *rhœsus*—were exclusively employed. After deep anæsthesia was produced a considerable extent of bone was removed from the left side of the skull posteriorly and laterally, so as to expose the occipital lobe and the greater part of the angular gyrus. The dura mater being reflected, the occipital lobe was cut off at the parieto-occipital fissure, and the tentorium-cerebelli was incised back to the lateral sinus. By means of small pieces of sponge the anterior folia of the cerebellum upon both sides were carefully and gradually displaced backwards, so as to expose the quadrigeminal bodies and the veins of Galen. With care the latter could be avoided, while the ganglia were destroyed by the galvano-cautery.

In one of the earlier experiments the occipital lobe was not removed, but it was found impossible to obtain a clear view of the ganglia without removal of the occipital lobe; so that this practice was resorted to in all the later experiments, for in order to limit the lesions to these bodies, it is expedient that they should be fully exposed. Bleeding from the veins of Galen is the most serious accident which may happen. The shock of the operation is considerable and stoppage of respiration was observed on several occasions when the cautery was being applied; but further reference will be made to this.

Experimental Observations.

Experiment 1. *Macacus rhœsus.*—Immediately after the operation, the knee-jerks were obtained equal upon the two

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sides and of normal range. The pupils were contracted to the size of pin-points, and the eyeballs were deviated to the left side.

On recovery from the shock of the operation the following observations were made: When placed on the floor the monkey hastened off to the side of the room, the gait being perfectly steady, not the slightest tendency to fall over or stagger being detected. There was no sprawling of the limbs nor were there any tremors. On one or two occasions when startled by a loud noise he was seen to fall, but rapidly recovered his stability.

There was clear evidence of hemiopia towards the right side. As regards hearing, when a bunch of keys was jingled to right or to left, a reflex movement of the corresponding auricle was observed. There was no impairment of ordinary cutaneous sensation.

Four days later the pupils were seen to be of normal size. The eyes were moved naturally either to right or left. There was no ptosis, nor any defect in the movements of the eyelids. As far as the observation can be relied upon when tested in a monkey, the pupillary light reaction was present. The gait was steady, climbing was easily accomplished and the grasps were good. The knee-jerks were natural.

During the first few days after the operation the monkey showed a slightly paretic condition of the legs, but this did not interfere either with walking or with climbing. It passed off after the first week.

During the month the animal lived after the operation, with the exception of the right-sided hemiopia, no symptoms were detected. He was well and active, and moved freely about the cage. He was killed under chloroform, one month after the infliction of the lesion.

Autopsy.—The left occipital lobe had been removed. The cerebellum was intact. The lesion of the quadrigeminal region was practically complete. The only portions remaining were the anterior border of the nates on the left side and the posterior margin—linear in extent—of the testes (fig. 1).

Microscopically.—Examination of the sections at the level

of the lesion showed that the ganglionic masses had been entirely removed, the line of demarcation passing through part of the Sylvian gray matter in the proximal parts and immediately dorsal to the posterior longitudinal bundles in the distal sections. The superior cerebellar peduncles, fillets, and internal geniculate bodies were intact.

Experiment 2. *Macacus rhœsus.*—Three-quarters of an hour after the operation the knee-jerks were obtained, but that on the left side was more active than the right. Both pupils were small, but not contracted to the size of pin-points; there was no deviation of the globes. The monkey was lively and able to move its limbs freely. He was able to raise himself without falling over or swaying. On the following day he was able to progress well, without any unsteadiness or tendency to fall. The usual tests showed that there was hemiopia towards the right side, and also a blunting of both tactile and painful sensation, and defect of localisation on the left arm and leg, and the left ear was also anæsthetic, as well as the left side of the face. There was no loss of motor power, as he was able to climb the lattice of the cage freely and to hold on tightly. There was no tremor on volitional effort.

It was difficult to be sure of the facts regarding hearing. No notice was taken of keys when jingled behind his back, nor was an auricular reflex obtained from either ear.

With the exception of a return to a medium size of the pupils—the left, however, remaining somewhat larger than the right—no change was detected in the symptoms during the ten days that the animal lived after the operation. At no period did he show any unsteadiness or ataxy.

Autopsy.—The superficial appearance of the lesion is shown in fig. 2. The left occipital lobe had been removed, and the cerebellum was intact. The lesion of the quadrigeminal bodies was the most complete of the series of experiments, no part of the superficial area of these structures being left.

Microscopically.—The sections showed that the lesion was very extensive, passing deeply into the tegmentum cruris on the right side. In this way there were destroyed

the tegmentum cruris, the mesial fillet, and the central auditory tract on that side. The nucleus ruber and superior cerebellar peduncle escaped. The nucleus of the third cranial nerve was intact. The right internal geniculate body was partly destroyed, but that on the left side was untouched.

In the distal or pontine sections the extent of destruction was the same as in the previous case, the line of the lesion passing immediately dorsal to the posterior longitudinal bundles.

Experiment 3. *Macacus sinicus.*—Within a quarter of an hour of the performance of the operation the monkey was able to sit up without support. The knee-jerks were equally and readily obtained. The pupils were contracted, but there was no deviation of the eyeballs.

On the following day one was unable to observe any evidence of instability of movement, the monkey sat up without any support and showed no tendency to fall even when incited to hurry. He was absolutely steady both when progressing along the floor and when climbing the lattice of the cage, and even when sitting upon a perch.

There was hemiopia towards the right side shown by the usual tests, and for one or two days after the operation there appeared to be some defect of sensation down the right side. There was no note upon the state of hearing. The monkey remained perfectly well for one month, when he was killed under chloroform.

Autopsy.—The left occipital lobe had been removed. The extent of superficial destruction of the quadrigeminal bodies is shown in fig. 3. From this it is evident that the region had been completely destroyed with the exception of a linear zone corresponding to the hind margin of the posterior bodies.

Microscopically.—In this case the sections showed that the ganglionic masses had not been entirely destroyed in the deeper parts, and that the adjacent and subjacent structures, fillets, superior peduncles and geniculate bodies were intact.

Experiment 4. *Macacus rhœsus.*—Immediately after the operation the knee-jerks were obtained, the left being some-

what more active than the right. There was also deviation of both eyeballs to the left, and the pupils were small and equal. Owing to a considerable amount of prostration, no observations upon the gait and other functions were made until the third day, when the following were observed.

By this time the knee-jerks were equal, the pupils of medium size, and the movements of the globes unimpaired. Instead of walking across the floor in a natural way the monkey was seen to pivot upon his buttocks, but there was no instability or falling, unless he were urged. The movements of the left arm and leg appeared defective, but this was due to the anæsthesia and analgesia, which extended over the whole of the left side of the body and limbs.

The appearance of the animal suggested a careful testing of vision; and the result showed that the sight was abolished on both sides. He took no notice of pieces of apple placed before him, or of a lighted taper brought in front of his eyes. He only picked up food when it was placed in contact with the right hand. No definite facts were ascertained regarding hearing. There was no response by blinking or starting when a noise was made near him, nor any auricular reflex when keys were jingled before his ears.

At the end of one week he remained in the same condition. He groped about the floor in an aimless way, evidently because he did not see at all. There was still left-sided anæsthesia, but no instability or tremors.

During the second week it appeared as if there were some vision. He took no notice of objects anywhere to the right, but pieces of apple when held immediately to the left side of the nose were seized. There was no evidence of vision in the outer parts of the left visual fields. Towards the end of this week slight but not constant tremors were observed in the left arm on volitional effort, such as were seen in cases in which a lateral cerebellar lobe had been removed. There was no further evidence of hearing and the left-sided anæsthesia remained as before. The monkey remained without any material change in this condition for one month after the operation. By this time, however, there was evidence that he seemed to hear, for a reflex was obtained

from both auricles when keys were rustled beside his ears, or when the hands were clapped in proximity to his head. The animal died six weeks after the operation.

Autopsy.—The left occipital lobe had been removed and the anterior part of the left cerebellar hemisphere was largely softened. On removing this the nature and extent of the lesion of the quadrigeminal region was apparent. This was complete, no trace of the bodies remaining. On the right side, however, the lesion extended forwards and outwards so as to invade the posterior part of the optic thalamus and the internal capsule.

Microscopically.—The sections proved the extension forwards of the lesion into the right pulvinar thalami and adjacent internal capsule. The right tegmentum cruris also was invaded, so as to involve the mesial fillet and the central auditory tract.

The explanation of the clinical phenomena in this case was therefore apparent, the blindness being due to the removal of the left occipital lobe combined with destruction of the optic radiations in the retrolenticular part of the right internal capsule and pulvinar thalami.

Experiment 5. *Macacus rhœsus.*—Immediately after the operation both pupils were found to be contracted to the size of pin-points, but after the lapse of about an hour became of normal size. There was deviation of both globes to the left side, the right eye having an inclination downwards, the left eye upwards. Occasional nystagmoid jerkings of the globes were also seen. All the ocular phenomena had passed away by the morning of the next day.

The knee-jerks were readily obtained and equal. On the next morning the monkey was again observed, when it was found that he could move his limbs freely, that there was no unsteadiness of gait nor any tendency to fall or to sprawling of the limbs. He sat quite steadily without swaying. Nothing definite, however, was made out as regards vision, hearing or common sensation.

Autopsy.—Fig. 4 shows the superficial extent of the lesion. This was incomplete inasmuch as the anterior and

posterior margins of both pairs were intact. On examining the sections through the intermediate or destroyed zone, it was seen that the deeper portions had not been completely destroyed. The Sylvian aqueduct had not been opened. The fillets, central auditory tract and tegmentum were intact.

Experiment 6. *Macacus rhæsus.*—The monkey recovered rapidly, and was found to move his limbs freely; when raised he fell backwards to the left side, and was quite unable to sit up without support. The left pupil was more contracted than the right, and there was a very slight conjugate deviation of both eyes to the left. The knee-jerks were not obtained.

On the following day the animal was well and active. When placed on the floor he lay on his belly, but when urged to move on rising he invariably fell to the left side, but there was no rotation. On volitional movement, *viz.*, when food was placed between the lips, there was tremor of the head, and if the left hand was brought up to the mouth, tremors were also observed in it. There was no limb palsy, as he held on firmly both with hands and feet. There was hemianopia to the right side, but no interference with cutaneous sensation.

At the end of a week the symptoms remained the same, the most obvious being a coarse tremor of the head and body on movement, such as taking food, having the characters seen in disseminated sclerosis in man. The movements of the right arm appeared to be steady, but when the left was brought into use the characteristic "intention tremor" was noted. The gait was unsteady. The knee-jerks remained in abeyance. Ten days after the operation the monkey presented the following appearances: The left arm was maintained in a flexed and adducted position, and a rhythmic tremor observed in it, even when the animal was apparently at rest. These movements occasionally ceased, but on attempting to lay hold of food they became wild and incoordinated. The gait was unsteady, with a tendency to fall to either side. The appearances presented by this monkey were in all respects comparable to those previously

seen in cases of cerebellar extirpation and lesion of the superior cerebellar peduncle.

The knee-jerks remained absent throughout. Hearing was not tested. The monkey died upon the twelfth day after the operation.

Autopsy.—In this case the left occipital lobe had not been removed. The lesion of the quadrigeminal region was incomplete. The left posterior body was completely destroyed, while that on the right side was only partially so. Both anterior bodies were intact (fig. 5). The left superior cerebellar peduncle, shortly before it entered the tegmentum pontis, was found to have been considerably injured. The tegmentum itself was not injured in any way. The microscopical sections corroborated the existence of a complete lesion of the left posterior body, while the superior cerebellar peduncle was found to be extensively injured.

Experiment 7. *Macacus rhœsus.*—In this case after the operation the pupils were found to be dilated and equal. There was a slight deviation of both eyes to the left, but no nystagmoid movements. The knee-jerks were present and equal. On the following day when more fully examined the monkey was observed to be able to walk, but tended to fall towards the right side. The gait was unsteady, and a sprawling action of the limbs was noticed. When sitting up, which he did without support, rocking movements of the body were observed. There was anæsthesia and analgesia down the right side of the body and limbs, and right-sided hemianopia. Two days after the operation well marked intention-tremors were detected in the left arm on volitional movement, such as picking up a piece of fruit, the right hand being used to steady the left. The swaying movements of the body persisted, but the general disturbances of locomotion were less. These appearances were observed until the death of the monkey on the seventh day after the operation.

Autopsy (fig. 6).—The bodies upon the left side both anterior and posterior, and the posterior body upon the right side, were found to have been destroyed, but the right

anterior body was intact. The lesion, however, had extended deeply into the tegmentum on the left side. This was so extensive as almost to consist of a hemisection, the left superior cerebellar peduncle and mesial fillet being implicated.

In two other experiments (8 and 9) immediate phenomena only were observed, as the animals did not live sufficiently long to permit of further investigation. In both the pupils were contracted to the size of pin-points, but there is no note of any deviation of the globes.

In both cases during the performance of the experiment, while the bodies were being destroyed by the cautery, respiration ceased, but was restored after an interval by the performance of artificial respiration. One lived 40 hours, the other 12 hours after the operation.

In experiment 8 both anterior and the left posterior bodies were destroyed, the right posterior being intact. In experiment 9 all the bodies were destroyed with the exception of the right anterior.

Analysis of the Phenomena following Destruction of the Quadrigeminal Bodies.

The pupils.—In all the experiments with the exception of No. 7 the pupils were, immediately after the operation, contracted to the size of pin-points. In the exceptional case they were dilated, but reference to the record will show that the lesion was less one of the quadrigeminal region than of the tegmentum of the pons. This pin-point contraction was only a temporary phenomenon, for in two days at most after the operation the pupils had returned to their natural size.

As regards the pupillary light re-action, in the cases in which it was tested it appeared to be present (so far as reliance may be placed on this re-action when tested in animals, where it is so difficult to eliminate the complication of convergence and accommodation).

Ocular movements.—In all the cases with the exception of Nos. 2 and 3 the globes were deviated to the left side.

This deviation was not of more than a few hours' duration. The explanation of this was not clear. The operations were all performed from the left side and with one exception the left occipital lobe was removed. Removal of this lobe, however, and the consequent hemianopsy is not sufficient to explain the deviation; nor does an unequal though bilateral destruction of the quadrigeminal region satisfactorily account for it, for it was absent in experiments 2 and 3 where the ablation was as complete as in Nos. 1 and 4, in which no deviation was noted. It was also observed in Nos. 6 and 7, where the lesion was incompletely bilateral.

It may be noted in this connection that a temporary deviation of the globes may likewise follow lesion of a cerebellar hemisphere or of the cerebellar peduncles, while we had previously observed it also in lesions limited to the tegmentum cruris.

As regards the ocular movements after the immediate phenomena had passed away, in no instance was ptosis or ophthalmoplegia detected. The absence of oculo-motor paralysis was accounted for by the fact that in no case was the lesion sufficiently deep or far forward to implicate the nucleus of the third cranial nerve.

Knee-jerks.—With the exception of No. 6 the knee-jerks were obtained throughout and equally on the two sides. In the exceptional instance they were not obtained at all, nor was any complication noted to account for their absence, with the possible exception that the monkey was in a condition of poor nutrition.

Station and locomotion.—It is in this connection that the most characteristic symptoms of gross disease of the quadrigeminal region in man are observed. We may therefore at the outset state that in the five uncomplicated cases of quadrigeminal ablation, in which the lesion was complete and bilateral, *no disturbance of stability or locomotion was observed from the time of the operation until death.* Monkeys with this lesion were able to move freely, climb the lattice work of the cage, and even to perch upon the ropes and bars.

In the two experiments (Nos. 6 and 7 in which con-

siderable instability was detected with sprawling of the limbs during progression, falling about and tremors of the limbs and body, adjacent structures were injured. In No. 6 the superior peduncle was implicated, and in No. 7 the lesion extended deeply into the tegmentum and involved the superior cerebellar peduncle and mesial fillet.

As might be inferred from the retention of equilibrium and the absence of instability no loss of motor power was detected. In every instance the monkey grasped firmly both with hands and feet. In experiment 1 what appeared to be a paresis of the legs was observed, but it was quite transitory and did not interfere with locomotion.

Sensation.—The observations upon the cutaneous sensation were variable. But it is evident that a lesion limited to the quadrigeminal bodies, as such, was not accompanied by any impairment of cutaneous sensibility, as in the three experiments, 1, 5 and 6, where the lesion was accurately defined. In No. 2 anæsthesia, analgesia and defect in the sense of localisation was observed on the left side. In this case these phenomena were due to an extensive implication of the tegmentum cruris on the right side. In experiment 4 there was complete left-sided anæsthesia and analgesia occasioned by the extension forwards of the lesion into the right internal capsule and optic thalamus; and in experiment 7 there was anæsthesia on the right side of the body from a lesion of the left tegmentum pontis.

These observations are in agreement with what has been noted in cases of disease of this region in man, namely, that *if sensory disturbances exist they are referable to implication of adjacent structures*, such as the internal capsule or tegmentum.

Vision.—Early in the investigation it was found necessary to remove an occipital lobe in order to sufficiently expose the quadrigeminal bodies. That the right-sided hemianopsy which was present in six out of the seven experiments arose from ablation of the left occipital lobe is obvious from the fact that no additional visual defect was produced by destruction, partial or complete, of the quadrigeminal region on both sides. In experiment 4,

where the monkey was rendered temporarily blind, reference to the record will show the co-existence of a left-sided hemianæsthesia, which with the left hemianopsia was due to an accidental lesion of the posterior part of the right internal capsule.

There is here therefore cogent evidence in support of the view *that destruction of these bodies has no direct influence upon vision*. If this were not so, the complete destruction which was effected in experiments 1, 2 and 3 ought to have caused total blindness.

Hearing.—Even under the most favourable conditions for testing, it is difficult to be certain as to the presence of deafness in an animal. Reliance in testing was placed upon the monkey's attention being arrested by sounds, such as the jingling of keys, at times when it was otherwise occupied, and by the presence or absence of the auricular reflex. Even in experiment 4, where the animal was blind, and the possibility of fallacy less likely, the state of hearing was uncertain.

In only one case to all the usual tests did the animal appear to be deaf, and in this instance subsequent detailed examination revealed an extensive and deep lesion of the tegmentum with implication of the central auditory tract. In experiment 1, where the lesion was accurately limited to the ganglia, there was no obvious evidence of deafness. From this it would seem that *lesion of the quadrigeminal bodies as such is not productive of impairment of hearing*, a fact which the anatomical connections of the posterior pair corroborates.

The voice.—There were no observations which led us to the belief that the voice or the emotions were affected by this lesion.

Respiration.—In two experiments, Nos. 8 and 9, in which detailed observations were not made owing to the short duration of life after the operation, respiration suddenly stopped at the time when the ganglia were being destroyed, even though the animals were well under the influence of the anæsthetic. On the employment of artificial methods the respiratory movements returned.

Conclusions.

The results of these experiments show quite clearly that destructive lesions *limited to the ganglionic masses* of the quadrigeminal region are not productive of any obvious permanent phenomena; for after the disappearance of some transitory symptoms, such as contraction of the pupils and deviation of the eyeballs, no residual effects are observed. Should such definite phenomena as instability of gait, tremors on volitional effort, anæthesiæ and defects of vision be obtained they are to be explained as the results of injury to adjacent and subjacent structures, such as the cerebellum, the superior cerebellar peduncle, the tegmentum cruris, the optic thalamus, the internal capsule, the optic radiations and the occipital lobe.

A short statement of the anatomical connections of these bodies so far as at present known, may help to explain the very subsidiary nature of their functions. Two chief sets of fibres pass to the superficial layers of the anterior pair, one from the occipital lobe reaching their destination by way of the optic radiations and the pulvinar thalami, while the other is derived from the optic tract. A few fibres from the mesial fillet appear also to enter the anterior pair. What the outgoing fibres of the anterior bodies are is not so clear. That fibres stream out and decussate in the "fountain decussation" seems certain, but the fact that there is no degeneration in the spinal cord after removal of the quadrigeminal bodies shows that descending efferent fibres do not pass directly into this structure.

The afferent fibres of the posterior pair are mainly derived from the accessory auditory ganglion of the opposite side crossing by way of the corpus trapezoides and entering the posterior pair through the lateral fillet. What has been described as the central auditory tract lies ventral to the posterior bodies as it passes upwards to the internal geniculate body, and has no direct connection with the posterior pair.

It is an interesting fact also that although the occipital corticifugal fibres terminate in the anterior bodies, no cor-

responding corticifugal fibres are found in the posterior bodies after ablation of the first and second temporal gyri.

It would therefore seem, when the anatomical connections are studied in association with the almost negative results of physiological extirpation, that the quadrigeminal bodies have only a very subsidiary relation to the special senses of sight and hearing.

It is difficult to understand the retention of irido-motor and general oculo-motor action after complete ablation, if the anterior bodies are the centres for co-ordinating visual impressions with the movements of the eyeballs. The only explanation would seem to be that the pupillary fibres of the optic nerve reach the third cranial nucleus by some other route than through the anterior quadrigeminal bodies, as indeed Bechterew has maintained.

We have attempted to account for the absence of deafness in strictly limited quadrigeminal lesions, by referring to the fact that the central auditory tract lies outside these ganglia, the posterior quadrigeminal body being merely an accessory structure and end station of the fibres of the lateral fillet.

The relatively small functional value of the quadrigeminal bodies in man and the monkey, as determined by these experiments, is in distinct contrast to their importance in the lower vertebrates, such as fishes, frogs and birds, assuming that the experimental lesions in these animals have been of a strictly limited character. The most likely explanation of this difference is to be sought for in the greater development and increased physiological value of the cerebral centres in the ascent of the vertebrate scale, and with this there is a corresponding diminution in their size both absolutely and relatively.

REFERENCES.

(1) Ferrier and Turner. *Phil. Trans.*, vol. 185 B., 1894.
Ferrier and Turner. *Phil. Trans.*, vol. 190 B., 1898.
(2) Longet. *Anat. et Phys. du Sys. Nerv.*, 1869.
(3) Flourens. *Récherches Experimentales, &c.*, 1824.
(4) Stefani. Referred by Sherrington. "Text-book of Physiology," Ed. by Schäfer, 1900.
(5) McKendrick. *Trans. Roy. Soc.*, Edin., 1873.

(6) FERRIER. "Functions of the Brain," 1886.
(7) BECHTEREW. *Arch. f. Physiol.*, 1884.
(7A) FANO. Referred by Sherrington. "Text-book of Physiology," Ed. by Schäfer, 1900.
(8) NOTHNAGEL. BRAIN, 1889.
(9) EISENLOHR. *Neur. Central.*, 1890.
(10) KNOLL. *Eckhard's Beiträge*, 1869, vol. iv.
(11) ADAMUK. *Central. f. d. med. Wissensch.*, 1870.
(12) SHERRINGTON. *Proc. Roy. Soc.*, 1896, vol. lx.
(13) CAYRADE. *Journ. de l'Anat. et Physiol.*, 1868.
(14) GOLTZ. *Funct. der Nervencentren*, 1869. 180.
(15) KOHTS. *Virchow's Archiv.*, 1876, vol. lxvii.
(16) ONODI. *Berl. Klin. Wochensch.*, 1894, p. 48.
(17) OTT. *Journ. of Nerv. and Ment. Dis.*, 1891.
(18) PRUS. *Wiener Klin. Wochensch.*, 1899. No. 45.

DESCRIPTION OF THE FIGURES.

FIG. 1.

Superficial appearance of the lesion in experiment 1. It will be noted that the whole of the superficies of the quadrigeminal region is destroyed, with the exception of a small portion of the left anterior body.

FIG. 2.

The lesion in experiment 2. In this case it extended deeply into the tegmentum.

FIG. 3.

The lesion from experiment 3, showing a complete superficial destruction.

FIG. 4.

The appearance of the lesion in experiment 4. This lesion is incomplete in so far as the anterior and posterior margins of the bodies are intact.

FIG. 5.

The appearance of the lesion in experiment 6. The destruction is practically limited to the posterior pair.

FIG. 6.

Represents the lesion in experiment 7. This is also incomplete in that the right anterior body is intact, the others being destroyed.

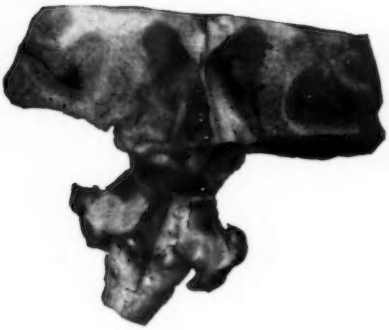

Fig. 1.

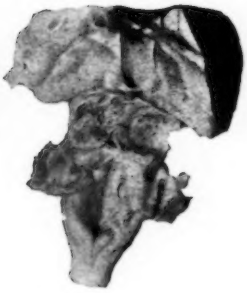

Fig. 2.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 6.

ON PARENCHYMATOUS SYSTEMIC DEGENERATIONS MAINLY IN THE CENTRAL NERVOUS SYSTEM.

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In a "Demonstration of various types of changes in the giant-cells of the paracentral lobules," a report of which, with illustrations, is published in the *American Journal of Insanity*, vol. liv., pp. 221 to 226, October, 1897, a description is given of the findings, in a case of senile melancholia, of "changes somewhat similar to those found in the motor cells of the cord and the medulla from which the nerve process has been cut off at the periphery." A type of this lesion is seen in the cells of the facial nucleus in a case of facial paralysis due to infiltration of the periosteum of the internal auditory canal, published in the *Journal of Experimental Medicine*, November, 1897. The lesion of the cells consists in a focal reduction of the size of the stainable portions, or complete decay into dust-like particles, leaving in certain places (usually opposite the axone) an area of an almost homogeneous transparent character, which progresses to general swelling of that part of the cell body, and usually to dislocation of the nucleus to the surface. In the above case of melancholia both paracentral lobules showed lesions of the large pyramidal cells. In all of them the stainable portions were smaller than normal. In part of the protoplasm there is in many cells a homogeneous dissolution of the structure as if the details had been washed out; the nucleus frequently seemed distorted and flattened, as if pressed to the wall by this swollen portion. Independent of this, or sometimes along the margin of the swollen area, there

appears an accumulation of yellowish pigment. Sections of other cells show practically no "glassy" swelling, but a peculiarity in having the very slender Nissl-bodies arranged in striking whirlpools, such as I have not seen in any normal cells, in fact, in no human nerve-cell outside of this case, and which, to my knowledge are not described anywhere. The nucleus forms in many cells the centre of these vortex formations, and frequently is so covered up by them that the Nissl stain does not give an outline of its capsule. In other cells there is a combination of glassy degeneration with whirlpool formation ; in some of them apparently two distinct foci of glassy decay separated by a distinct capsule of slender Nissl-bodies. An explanation of this condition is somewhat difficult. It might be intelligible from the supposition that the glassy degeneration of part of the cell body might produce certain distortion of the internal structure in the rest, as is actually seen in many of the cells, and that cells which showed these vortex formations, would be in a state of recuperation. I would not like to be understood as considering this case as the type of what occurs in senile melancholia, since other experience in this line is not favourable to this view, and since a further examination of the basal ganglia will probably reveal lesions of the projection fibres within the internal capsules. During the correction of the proof, the following foot-note was added : "This proved to be true to a limited extent only. A second case, examined later, a patient with typical senile melancholia, gave evidence of identically the same changes as those described above. Both will be published in full as soon as the basal ganglia of the second case are examined."

During a period of three years eight cases of this type have come to autopsy. They have greatly helped to clear also the clinical side of the picture. It seems opportune to publish them at the present time, especially since publications are coming forth which in part belong to the same field, and may be furthered by a communication of our findings.

The following notes are abstracts of the histories of my cases, given rather fully, in order to allow the alienist to

form an opinion of the varying mental picture, as far at least as our records furnished the data. The relatively atypical nature of most of the cases will excuse the length of some of the records, without which it would be impossible to appreciate the peculiar similarity with, and yet difference from, general paralysis. To the record of my cases I add further an unpublished case of Dr. August Hoch, and abstracts of other cases collected from the literature.

Case 1 (from Dr. Noble's service).—*Psychosis beginning like an ordinary climacteric melancholia, then passing into a condition of mutism with vague nervous disorders following a slight left hemiplegia. Three weeks before death diarrhœa; finally twitching of arms. Aged* 68 *years.*

D. S. R., born in 1829, widow, housewife. The patient's grandmother, father, two brothers and one son were insane. October, 1895, she became depressed; "she groans, wrings her hands, sways from side to side in mental anguish, worries in self-reproach for having murdered a whole family." She became sleepless, restless and noisy, refused food, and was admitted to the hospital on the eleventh day of her illness. She was fed with the tube for several days; in November she ate well; only occasionally till December, 1895, she required tube feeding; her mental condition remained about the same. She would say that she was the devil, was much depressed, rarely answered questions, and was quite inactive. March, 1896, she stayed in bed; later, during the summer of 1896, she was up during the day. She hardly ever spoke, and did not answer questions, and remained depressed and inactive, and lost flesh. October, 1896, the first entry concerning her physical condition was made. There was no evidence of dementia, but complete inactivity and mutism. The patient was always in bed. The state of nutrition was fair, but the face pale; the head large, rectangular, prominent in front; the pupils responded sluggishly to light; exaggeration of the knee-jerk is noted and ankle clonus on the left side. No reaction to prick of pin. The sleep was poor. In December, 1896, the tendon reflexes were less active; almost no knee-jerk was elicited on the right side. Bed sores began to form. January, 1897, she answered a few questions, but sometimes she did not open her eyes when spoken to, and for several days she did not swallow readily, but held the food in her mouth for a long time. This was transitory. From the answers no evidence of real deterioration was gained: There was then

dribbling of urine and consequent eczema; the left arm and leg are held rather rigid, and the knee-jerk is moderate on both sides, especially on the left, on account of the rigidity. Patient takes hold of the hand, but is slow in her motions. January 25, she is said to take nourishment better and to converse somewhat more freely; "she told me this morning that she was frightened, and that she heard noises about her." End of February and in the beginning of March occasional diarrhœa was reported. March 16, 1897, twitchings of both hands and feet were noticed, and a tendency to contraction of forearms and hands, the right pupil was wider than the left; the pulse 138, weak; the respiration 40, and the temperature 100·3°. The patient was uneasy when put on her right side, and required to be moved back to the left. No definite picture of the mental condition could be made on account of the continued mutism. Death occurred March 17, 1897.

The following is the summary of the autopsy report:—

Subcutaneous ecchymoses on the forearms and over the ensiform process. Sacral decubitus. The *heart* rather large, with atheromatous patches on the mitral valve and on the beginning of the aorta. The *kidneys* slightly granular, with a number of small cysts, and sclerosed parts in the right lower pole. Weights 84 and 87 grammes. The *lungs*, with slight adhesion of the right apex, slight bronchitis in the right lower lobe, probably with beginning broncho-pneumonia. Weight of the left lung 265 grammes, of the right lung 360 grammes. Remnants of parametritis (strings of calcareous nodules). One large gall stone. *Skull*, with vortex-like *hyperostosis* on the inner plate of the frontal bone, thick. The calvaria weighs 610 grammes, instead of about 300 grammes. *Pia* œdematous, thickened and white along the longitudinal fissure. *Basal vessels* whitish. *Convolutions* somewhat atrophic.

No macroscopic lesions in the brain, except a *small scar in the right caudate nucleus*, nearly reaching the internal capsule. The spinal cord was not removed. The microscopic examination furnishes the following additional data:—

The specimen from the lung showed a number of alveoli filled with blood and with polynuclear cells, and the kidneys typical arterio-sclerotic shrinkage, with marked sclerosis of many glomeruli and their capsules. The microscopic examination of the various parts of the cortex (right and left paracentral lobules, F_3, T_1, and cornu ammonis) showed no tissue alteration, except the very marked axonal reaction of the Betz-cell on both sides,

described in the first communication, and a small hæmorrhage in the cornu ammonis. The semilunar ganglion showed advanced pigmentation of almost all the cells; in many cells the nuclei are dark, in various stages of homogenisation, in others clear and large, and in a few eccentric. The stainable portions are very scanty. Sections through the basal ganglia showed that the internal capsule was not involved in the scar.

Case 2 (from Dr. Boynton's service).—*Senile depressive hypochondriacal delirium with profound emaciation and aimless restlessness. Rapid physical decay without typical nervous phenomena. Death from broncho-pneumonia. Duration of the whole process two months. Aged* 63 *years.*

J. W. H., born in 1834, shoemaker, married. His father died of apoplexy. The patient was a very conscientious worker. In 1888 he fell over unconscious (fainting?) when walking across a bridge, and was sick for two days; in 1894 he had a "fainting turn, a cold sweat came over his chest and he smelt like a dead person." Within the last two years mental efforts have become more difficult to him (such as speaking in his "lodge"); his son noticed forgetfulness for three or four years. Last winter (1896 to 1897) the patient had an attack of influenza from which he recovered imperfectly. In June, 1897, a "specialist" found his spleen enlarged. After three days of medication he became delirious; he said he was filled with wires. Reduction of the amount of medicine left him with the same somatic delusions. His wife found him a few days later out in the back yard, shoes and stockings off, carefully looking down into the toe of the stocking. "The whole of my insides have fallen out; I am trying to find them down there." In August a neighbour said something about a thing smelling bad, and the patient began to have ideas that *he* smelt bad, that he was all rotten; he said of the electric cars, "see how fast they go by now, they all smell me;" passers-by "held their handkerchiefs to their noses or went on the other side." Or he would get up at night saying, "my legs have rotted off and gone and I want to see if I can walk." He refused food because it went "to the top of his head," and on August 27 made several weak attempts at suicide by means of a twisted cloth, a shoe knife, and oxalic acid. "It is no use to live any longer." At the same time he became confused, had difficulty in recognising neighbours, was restless and did not sleep. When admitted, August 31, 1897, he only weighed 100 lbs. against 156 lbs. one year previous, and he had tremor of the hands and

feet, arterio-sclerosis, prominent veins, slight enlargement of the heart, exaggerated tendon reflexes, sluggish pupils and arcus senilis. He was dazed, restlessly and helplessly standing or wandering without aims, occasionally mumbling unintelligibly; he once showed marked fear of being killed, and then could apparently not answer questions or even understand them; he did not show the tongue even when shown what was wanted. He was tube-fed from the beginning. He usually stared in a vacant manner, mouth open; once answered to the question who he was, "it was H." From September 3 he was kept in bed; restless, pulling at the bed-clothes, probably utterly disoriented. September 13: his respiration was 38, the temperature 103·8°. Hypostatic pneumonia. Death at 6 p.m.

Anatomical Summary.

Arterio-sclerotic kidneys, practically no other internal arterio-sclerosis. Heart weighing 280 grammes; flabby, but few atheromatous patches at beginning of aorta. Slight old adhesion of left lung; hypostasis of both lower lobes. Skull thin; dura adherent with small endothelioma in the temporal fossa; pia slightly thickened and œdematous, slightly whitened, with arachnoid cysts; nowhere adherent. A small lipoma in the pia of the tuber cinereum. Careful inspection of the brain revealed no macroscopic lesion in any part. No granulations of ependyma.

The cortex of the *paracentral lobules* shows the typical axonal alteration in most of the largest pyramids (Betz-cells). Among fifty there is only one normal, a few have whirlpools. The large pyramids of the third layer are not affected, except that they frequently show a decided rarefication of the stainable spindles in parts of the apex and around the perfectly normal nucleus; especially in the polymorphous layer, many nuclei are folded. In the second and third layers there are rather numerous pigmented glia cells, and a moderate number of satellites in the deeper strata. No spider-cells. The nerve-cells are moderately pigmented.

The *frontal cortex* also presents the axonal reaction in many of the largest elements. The cells are generally more pigmented and with a spongy appearance of the protoplasm; the nuclei are, however, normal. The number of satellites is a little greater than in the paracentral lobules.

The *cornu ammonis* and the *cerebellum* are normal; in the *Golgi specimens* the dendrites have a normal number of gemmules. The cells of the motor plate of the upper *cervical cord* are con-

siderably pigmented, and many of them small and with few lumps of stainable substance. There are two instances of axonal degeneration among the twenty cells examined ; four are normal and large, two are mainly pigment bags. In several cells the lining of the nucleus is denser and its granules larger and the nuclear membrane is lost. Among the long spindle-shaped cells of the centre many have thick spindles; but one shows the axonal reaction, also one of the large cells of the dorsal horns.

The cells of the *semilunar ganglion* are of varying type; pigmentation is more moderate than in case 1, but present in most cells ; the stainable substance is either present in rather large lumps uniformly throughout the cell, or much more frequently there is a marginal row of large lumps while the central part of the cell has smaller granules. In many cells the central granules are more or less hazy or even quite indistinct and the marginal lumps clotted together, and sometimes such clots of lumps form caps around the nucleus. There is probably no typical axonal reaction. The nucleus is frequently considerably eccentric ; but nowhere does it break through the marginal ring of the lumps. The nucleus is normal in many cells, in others the net of granules is more dense, and all stages can be followed into the type with dark sap and absence of membrane.

In the left *lung* pneumonia with hepatisation and the diplococcus pneumoniæ is established. The *kidneys* show only moderate arterio-sclerosis. The *liver* is hyperæmic and the cells moderately vacuolised.

Case 3 (from Dr. Fleming's service).—*K. H., senile deterioration, with depression, progressing in the course of three to five years to delusions of persecution and restlessness, and finally disorientation ; arterio-sclerosis. Peculiar attacks of tremor, January,* 1897 ; *from October,* 1897, *progressive exhaustion. Death from pneumonia, December* 18, 1897. *Age between* 62 *and* 71.

Patient is a woman 61 years old (her niece says about 70). The father died of phthisis and the mother in the latter years of her life developed a mild form of insanity. The patient was born in Ireland. She was always healthy ; came to America in 1867 and worked as a domestic. Nothing is known of her sexual history, nor is there any statement concerning alcoholism. She had no school education, can neither read nor write.

Five years ago she began to show loss of memory and to worry over trifles ; she was depressed, cried frequently, became retrospective ; she talked and cried over things that had occurred

in her early life. Two years ago she gave up work and lived alone; when her niece would visit her she would find her with but little to eat, but the patient would say that she did not want anything. Delusions developed at this time, that people were stealing and wearing her clothing. About a month before admission she boarded out, and the lady with whom she boarded was unable to keep her in the house—she would get up in the night, wander around the house, and during the day she would try to get away, saying she wanted to visit the neighbours. She became disoriented; did not realise that she was in W———, but thought she still lived in G———. (She had formerly lived there.)

She was admitted July 25, 1896, because she began to wander away from home aimlessly, and got lost. Since admission she was somewhat depressed—cried when spoken to. She removed her clothing and at times tore it, and was very restless, and her general intelligence was very defective. No delusions or hallucinations were elicited, the patient often talking to herself in an incoherent manner.

Physically she was very poorly nourished: the pupils sluggish, the knee-jerks diminished. General tremor was present at times, the grasp somewhat weakened. Dulness at the apex of right lung, without râles, but with prolonged expiration. A systolic murmur partly replaces the first sound in the mitral area. Atheroma of temporals and radials. Inguinal hernia, more marked on the right than on the left.

By August, 1896, the patient did not cry as much as formerly, answered when spoken to, but could not give a coherent account of herself. In December, 1896, she became more restless, undressing herself often, and tearing her clothing. January 19, 1897, she was unusually restless and destructive; about 11 a.m. she began to tremble all over; her mouth was drawn to the left and she was unable to speak for ten minutes; about 3 p.m. there was a similar attack, lasting only a few minutes. Nothing of the kind was observed later.

Towards May the patient became more feeble, unable to stay up long, but very restless in bed. In June, œdema of the eyelids was noticed once; but the urine was found normal on repeated examination. In July she was stronger and up; it was impossible to keep her in bed; but in October she became finally bedridden. The physical examination brought out nothing new, except that the left knee-jerk was a little more marked. In December, 1897, œdema of the feet appeared and occasional

diarrhœa; the restlessness continued, the hernias became more troublesome, and finally, December 24, 1897, the patient succumbed to a pneumonia.

The autopsy revealed general atrophy, pneumonic infiltration of the posterior part of the right lower lobe and moderate œdema of both lower lobes. The anterior surface of the right lung and both apices showed old pleuritic adhesions. The heart had very brittle and tortuous coronary arteries and there were thickened patches in the mitral and the beginning of the aorta; small, slightly cystic kidneys, partial sclerosis of the basal arteries of the brain, a few hyperostoses of the frontal bone. The brain showed nothing, except general atrophy (1,132 grammes) and œdema of the pia; no granulations of the ventricles. The other organs weighed: the spleen 40 grammes, the kidneys 70 and 75 grammes, the heart 140 grammes, the right lung 330 grammes and the left 335 grammes, and the liver 640 grammes.

Microscopically we found a pneumonia with Friedländer's bacillus, and in the cortex marked shrinkage (the œdema alteration of Hoch) and axonal alteration of the Betz-cells on both sides, also of a few of the larger cells in the first frontal gyri, but none in the temporal, third frontal, cornu ammonis or cerebellum. The sympathetic cells, however, showed much pigmentation, and almost throughout the sections a condition similar to that in case 6.

Case 4 (from Dr. Gurley's service).—*Depressive hallucinosis; after a six months' course rapid decay and fatal termination within a few weeks with features not unlike those of general paralysis, but without speech disorder or other distinct nervous symptoms. Aged* 50 *years.*

F. P. G., born in 1849, painter and agent for sewing machines. The maternal grandmother was insane after the birth of patient's mother and died insane. The patient reports of himself excessive masturbation from 10 to 12; at 15 consequent nervous debility, dizziness, inability to bear heat of the sun, two "sunstrokes" (symptoms vague); no full recovery although he was sometimes better, sometimes worse; at 20 to 25 a course of books and medicines on lost manhood, &c.; at 23 he married, and two to three months later was divorced; re-married at 25; masturbation during marriage; probably no syphilis; he was always (at least since marriage) melancholy, easily disturbed; it was hard to get him up after an illness, especially since 1894, when he had "lead poisoning" (continual

constipation, pain throughout the intestines, arms, and legs, swelling of legs); he was also subject to bronchitis, and since marriage to "liver trouble" (attacks with coated tongue, retching, vomiting, and a soreness in the hepatic region). Since 1894, a change of character became noticeable, a disposition to let things drift and to spend more than he could pay for, gradually increasing to a maximum in the spring of 1898, when he bought boats, thinking he could make plenty of money. He could not pay for them and failed; he did not worry about his work; he became interested in socialistic papers and at times excited. He grew more secret and quick to take offence, would not be dictated to by his family, his wife did not dare to ask about his business. In the spring of 1898, on return from a seven weeks' trip, his eyes looked heavier; October 10 he had abdominal pains, he was fearfully depressed, thought he could not get well; that there were people around who were going to harm him; and that the doctors were going to cut him up; he thought his wife and children were against him. He was in bed until admission. From November 5 he had hallucinations of hearing: He heard his wife tell their daughter of 17 and his physician that he had laid on her too much, whereupon the doctor said that such a mean man as that should not be allowed to live; and the daughter said she did not know that he was such a mean man. Later he heard them say such a mean man ought to be killed, and they talked about having the nurse kill him. He did not believe the improbability of such conversation; he must believe it because he heard it. He remarked it was all right, and that he ought to be killed. He produced self-accusations, mainly for trivial real deeds, as thefts at 12 and 13 of money from an aunt, and his behaviour with his wife. The voices became very numerous and distressing by their threatening or disgusting contents, as "God is a whoremaster, and all the world is a whoremaster; come to meet your doom." On November 8 the patient cut his throat to forestall others harming him; November 15 he hit himself over the head with an iron; he was very changeable, at times apathetic and listless, then suddenly alarmed, or at times in deep study and averse to conversation, at other times talkative. But orientation was never affected. November 15, 1898, on admission, he was carried into the ward. "The arrest took all the strength out of my legs." He soon rallied. From the beginning he was constantly under the influence of hallucinations. Far away voices say all kinds of abominable things like "God is a fool: Frank, come here, I want you, arise, turn

to the left and meet your doom," &c. He does not answer the voices but obeys them in all his actions (he took off his shirt several times, looked into the mirror to see whether he really was a beast, &c.), and frequently is at a loss: "I don't know what to do; one tells me to go up to the right, and when I did that was not right; others tell me to turn to the left, and when I did it that was not right. I don't know what to do; I wish somebody would tell me straight." Sometimes the voices are just ideas, then they become faintly audible and then louder, as if from the corridor.

The patient is a man slightly below medium height, weighing 120 pounds, with gray hair, anæmic mucous membranes and blue sclera, slight arcus senilis, but no atheroma. The wound from his attempt at cutting his throat is healing. Apart from muffled heart-sounds and a systolic murmur over the apex and left axilla and subclavicular fossa, disappearing in recumbent position, he shows much exaggeration of the knee-jerks, less of the elbow reflexes and the cutaneous reflexes, a slight fine tremor of the hands and fingers on rest and rotation, and some defect of coordination of fingers with eyes closed. Gait and speech were normal, sleep and appetite fair. The patient often sat by himself with a perplexed and depressed look; he gradually (during December) began to associate himself as travelling companion to any new and restless patient, walking up and down with him. His fear of execution varied. A voice told him to go out of doors with the others to have his bowels taken out. "I am ready to meet whatever is coming; the voices tell me my head will be cut off, the eyes taken out, the teeth pulled. I expect something dreadful is going to happen." Or he heard he was never married, the minister not ordained and the children not his, his wife dead, &c. When she called he thought she was a little stouter and her voice a little different and he doubted whether it was she. In January he appeared more indifferent to the voices and more and more devoid of affect. Orientation was always good and he showed no deterioration of memory; he had some sort of insight—"my mind is in a bad state; it keeps on thinking bad things; and I hear voices calling me to my doom." He often came to the physician with the question: "Did you call me? A voice told me to come to my doom." He took a scratch from an excited patient without resistance, "it is part of my doom." He is almost over willing to obey any instructions or orders, just as he does with the voices. Some of the voices he attributes to his own mind, and January 5, 1899, he remarked that the people around

him must read his thoughts, because as soon as he thought of anything he heard it said after him. The self-accusations are meagre and are overshadowed by hallucinatory incriminations against which he remonstrates, such as having killed someone: he had only let his mother-in-law's nurse go. The idea of punishment increased. January 24 he bared his buttocks and deliberately burned them against the radiator, saying he wanted to begin on his punishment. In March, he often asked for his punishment. One day he said the food tasted metallic and he was constipated: "part of my punishment." Hæmatoma of the ears developed in April (attributed to the watchman); then he attempted to gouge his own eyes, because the voice told him to and because he was mixed up (he was ashamed of his actions); when his hand had to be restrained he tried to strike his eyes on sharp corners of chairs, faucets, &c.; he often got up at night obeying the "rise and meet your doom." Under increasing emaciation the patient finally showed a few times subfebrile temperature and vomiting, and he died unexpectedly in a collapse on May 18, 1899, after having stayed in bed four days.

The *autopsy* was made twelve hours after death and established general emaciation, residuals of hæmatoma of both ears, decensus of transverse colon and stomach, a few cysts of the kidneys, moderate adhesion of the dura to the skull, œdema and slight milkiness of the pia; the lobes were easily separated. No granulations of the ventricles. No evidence of atheroma, not even in the beginning of the aorta. General atrophy of the organs; heart 170 grammes, left lung 200 grammes, right lung 210 grammes, liver 820 grammes, spleen 50 grammes, right kidney 85 grammes, left kidney 75 grammes, brain *in toto* 1,285 grammes.

The *paracentral lobules* show no tissue alteration characteristic of general paralysis, but the axonal reaction in the greater number of the cells, in various degrees and combined with rather extensive pigmentation in many of the cells. Around the smaller cells of the deeper layers there are frequently a group of satellites. The *frontal* and *cerebellar cortex* are free.

Sympathetic (semilunar) ganglion. About one-fourth of the cells are fairly normal (large peripheral lumps and small central ones of stainable substance, and a fairly central nucleus) the majority of the cells have only a peripheral crust of stainable substance, not in distinct lumps. The centre is glassy, only occasionally laden with pigment granules, and the nucleus is more or less eccentric, in some cells reaching the surface completely as in the pictures of pronounced axonal reaction. Only few nuclei show

degrees of homogenisation and slight loss of the membrane. About half the cells show pigment, but not to excess. There are no eosinophilous granules.

The *Gasserian ganglia* have large cells with fairly even and dense distribution of medium-sized and small stainable particles and rather numerous "fatigue nuclei" (condensation and beginning loss of membrane).

Case 5 (from Dr. Gurley's service).—*Alcoholico-senile deterioration. For two years periods of one to three weeks of weakness and indistinct speech. June*, 1898, *jerking of all the muscles and acute hallucinatory delirium, followed by a partial remission; from September*, 1898, *depressive delirium with somatic delusions and increasing weakness. Death from broncho-pneumonia, October* 28, 1898. *Aged* 57 *years.*

T. D., 57 years old, teamster, with good family history as far as known; with uneventful personal history, except for excessive alcoholism; he always was a heavy drinker, of whisky mostly, often taking as much as a pint daily, and he was frequently intoxicated. He married at 20, and had seven children; four died in infancy. He had not worked steadily for two years. In the summer of 1897, while working in the yard, he fell, not entirely unconscious, but with great weakness, so that he could not use either leg; understood what was said, but could not speak; he made a complete recovery in three weeks. However, from this time he had a monthly occurrence of attacks of the same kind, weakness and indistinct speech; he could not get upstairs without aid, and it was difficult to understand him. These attacks lasted one week. From the first attack the patient showed more irritability, he was hard to get along with, and drank much. June, 1898, he had jerking of all the muscles, so that he could not stand up; he acted strangely, and showed slight asymbolia, *e.g.*, he held his pipe in his hand and tried to cut it as if it were tobacco; then, about June 14, his speech became very thick and indistinct, yet he was jabbering all the time; by June 20 he had to go to bed, where he was unable to move without assistance (?). The following day there was a sudden appearance of hallucinations of sight and hearing—fairies and witches and people about the house; carpenters boring holes through the walls. He jumped out of bed, dressed, and went into the street, consulted lawyers about papers he imagined he had lost, imagined people were trying to get his property, &c. His gait was feeble and shuffling, his speech silly, careless and

at times unintelligible, his attitude somewhat bewildered. He was admitted July 1, 1898. The following is an abstract of the *status* of July 9:—

Very gray thin hair, grayish moustache and beard; fair nutrition, flabby muscles, eczema on chest. Slight hypertrophy of the left ventricle. Hard radials. Marked arcus sensilis, internal strabismus of right eye (from childhood), chronic conjunctivitis. Hearing very defective (watch not heard in contact); odour of oils not recognised. Tactile sense not acute. *The knee-jerks were absent*, the reflexes of the arm present. Slight tremor of hand and tongue. Bleeding hæmorrhoids. The writing tremulous and defective in spelling, not unlike that of a general paralytic, whereas the speech was merely careless. The patient was quiet and tidy in the ward, occasionally wandering restlessly, with a simple apathetic manner, and slow, somewhat unsteady gait. He was very deaf, and slow in answering, and at times rambling. Place and persons were recognised; time orientation defective—year, month, hour of the day, age, &c. Only the simplest calculations were done; but he has insight into his difficulty and in the defect of memory. Patient has a vague idea that he sometimes was pushed about—"They keep me here wrestling and I don't know what they do it for, because I am no fighting man. I have no fault to find with the folks here, only they have a good time, I suppose." His son-in-law "stole his will." He admits insight into the hallucinations at home; he hears and sees "nothing imaginary now," and "feels very silly." He ascribes his failing to drinking. No history of syphilis could be obtained.

From August 19 his appetite failed. September 15 everyone called his name at the meals. From October 4 he became restless at night, pounding the door, seeing potatoes, grass, roads, a quarry on the floor; praying. From October 11 he groaned also by day, praying, "Oh God, Oh God, Oh! I am in hell, pretty near." October 12 he jumped into a shallow pool of water on the lawn, and when his daughters visited him he prayed for their salvation, making a scene and crying bitterly. October 15, he "wanted to lick all the devils in hell, out of hell, and below hell." He became unsteady, stumbled and fell around the ward often. October 17, he was leaning over the side of his bed, coughing and retching: "The devils are into me and I am trying to get them out." After October 20, he also caused a prolapse of the rectum. "The devils have gotten down there and I try to get them out." He tore the mucous membrane, and bled. Finally, October 22,

a rise of temperature and signs of broncho-pneumonia came on, and the patient died October 28.

The *autopsy* established broncho-pneumonia and œdema of both lower lobes, the left lung weighing 555 grammes, the right 830 grammes; a milk-spot on the anterior wall of the right ventricle and hypertrophy of the left ventricle (the heart weighed 365 grammes), with a few yellow patches in the mitral valve and a very few atheromatous elevations in the beginning of the aorta; a small cyst and a small scar in the right kidney (right kidney 112, left kidney 125 grammes); slight thickening of the capsule of the liver (1,470 grammes); distension of the stomach and intestine, hyperæmia of the rectum and a healing ulcer of its mucous membrane; atrophy of testicles. The skull was normal, also the dura; the pia was moderately œdematous and very slightly hazy over the blood-vessels, perfectly translucent at the base and thin over the Sylvian fossa; the lobes were easily separated; on the right anterior cerebral artery there was an atheromatous patch; the two sixth nerves were of equal size. No granulation of the ventricles. Weight of the entire brain, 1,235 grammes. The dissection of the formaline-hardened brain into the thin laminæ gave no abnormal findings, except perhaps very slight broadening of the vessels and perivascular spaces in the external capsules.

The cortex of the first frontal and paracentral lobules were examined with methylene blue after alcohol fixation. In the paracentral lobules the tissue as a whole has a normal orientation of the cells, no marked vascularity, a rather striking abundance of pigmented spindles with poorly defined nuclei in the adventitia of some medium-sized vessels, but no characteristic infiltration of the sheaths, and a moderate increase of "free nuclei," many of which surrounded with slightly pigmented protoplasm, either free in the ground substance or as satellites, and about as numerous in the layer of the medium-sized pyramids as in the polymorphous layer, certainly not more numerous around the affected cells. The first glance at the specimen shows the axonal reaction of the Betz-cell, with almost no exception and in various degrees, and modified by various degrees of pigmentation. The larger and medium-sized pyramids are not altered in the same way, but show very marked rarefication of the stainable substance of the apex process; and many of these cells and especially the polymorphous cells are pigmented and their nucleus is shrunken a little.

In the frontal cortex there are but few cells with axonal

reaction; many pyramids have a very slender rarefied apex and pigmentation is prevalent. The cerebellar tissue is not altered. The *Purkinje-cells* have no axonal alteration; the spindles are plain and often form caps around the frequently irregular nucleus. In the spinal cord the *motor cells* are mostly normal, though perhaps a little pale and much pigmented; the nuclei are darkened a little in a few cells, and very few show the typical axonal alteration. The cerebellar afferent cells of *Clarke's column* are all affected and show uniformly the axonal alteration, especially well on longitudinal sections. There is a moderate number of corpora amylacea throughout the cord, but no evidence of posterior sclerosis. The cells of the *spinal ganglia* are large; not over one-third of them are pigmented. The nucleus of all the cells is without membrane, more or less dark, granular, of uneven slightly irregular outline, but with a plain nucleolus. There is a halo around the nucleus. The stainable substance of the cell body is rather heavy along the periphery, and thin or absent around the nucleus. A few cells show the axonal reaction. Among the internal organs, the *liver* shows a very slight increase of connective tissue; the *lungs* a moderately extensive infiltration with rich infection, with streptococci and staphylococci, and the *testis* has unusually open canals, but a fair amount of sperma in various stages of formation. The *kidneys* have but few obliterated glomeruli with thickened capsule; they are on the whole well preserved. In one papilla of the kidney there is a small curly fibroma well limited against the other tissue. The kidney has a few septa proceeding from the capsule and rather thick vessels, but few sclerosed glomeruli. The liver has not much blood and a moderate fatty vacuolisation of the cells in the periphery of the lobules.

Case 6.—*Recurrent* (*partly hysterical and hypochondriacal*) *depression, with several exacerbations and partial remissions; termination in stupor with some twitching, and death by pulmonary embolism. Aged* 44 *years.*

T. J. H., born 1856, painter, married. Family history: mother died of brain fever; three of the patient's cousins have been insane, also his paternal aunt, and one paternal uncle is a drunkard; a sister is melancholy. They are described as a rather nervous, excitable family. The patient at the age of 9 months injured his right hip and leg, which remained short. At the age of 14 years (1870) the left knee was crushed by a

waggon. The patient went to school up to 1871, "never very bright," from childhood he was quarrelsome and cranky, and he often picked up imaginary insults. He did not get along well with his step-mother. From 1871 to 1874 he turned a wheel in a rope factory, and the next seven years he worked in a pottery. In 1878 the patient was troubled with sleeplessness and melancholia. In 1879, he was at the Carney Hospital with "symptoms of paralysis and severe 'convulsions,' and exaggerated shaking with apparent loss of consciousness and loud outcries." One night he got up from this "unconsciousness" to look in the mirror; he denied this at the time, but admitted it later. He was admitted to the *Danvers* Lunatic Hospital, September 19, 1879 ; he ate and slept well, was hysterical and lied a great deal, telling many foolish and impossible stories, and he had a series of convulsions relieved by an active cathartic. From February 20, 1880, he became more cheerful and contented, gained in weight, and was discharged *recovered* March 12, 1880.

The patient returned to his work, which he changed for house painting; this he continued up to April, 1898.

In 1889 he married, after an uneventful life of sexual abstinence, a woman of doubtful character. In 1892 or 1893 he acquired a urethritis from her; otherwise he knows of no sexual disorder, except that he failed to have erections from the Spring of 1898, while before he had cohabited about twice a week. He also mentions a lead colic in 1893, which kept him two weeks in bed. In 1896 the patient had an operation for rectal fistula. About that time he did not sleep well, and took sleeping medicine regularly since that time. He was afraid of getting sick and unable to make a living; finally, April 18, 1898, he quit work on account of depression, shaking of hands, and a pain in his bowels and lacking ambition. He became hypochondriacal, went from physician to physician, exaggerated his symptoms (*e.g.* his sleeplessness). After taking an overdose of cathartic pills in October, he began to feel that his bowels were ruptured. He was nervous, much excited, and on October 28 he had a marked convulsion of epileptic character. At home he was threatened of being strapped down for being so restless, whereupon he *cut his throat* with a razor, and was admitted here *November* 1, 1898, "for having complained of pain in his bowels, crying hysterically and for having attempted suicide."

Here the patient appeared physically normal, but for his traumatic deformities of both legs, and hypochondriacal exaggerations and prolix reiteration of his ailings, the too active motion of

his bowels, and the wound of the neck, &c., the circulation in his legs—"you see, doctor, my legs are rotting off," when they were slightly blue and cold. "You will see I am going to die a terrible death."

At night the patient was frequently restless, moaning, out of bed, and requiring hypnotics.

A careful physical examination established, besides his deformities and the healing wound, exhaustion, cold extremities, a numb feeling of the legs with somewhat sluggish tactile reaction, some epigastric pulsation. The knee-jerks were equal, not exaggerated; he had a fine gelatinous tremor of the tongue, and a coarse jerky tremor of the hands at rest, increased on motion. The abdomen was scaphoid; the internal organs and the urine normal. He improved somewhat physically and also mentally, when, April 24, 1899, he was taken with a febrile tonsillitis. During the night of April 25-26 he became greatly agitated and noisy, very fearful of being killed, burned, &c. In the morning he had wild, staring eyes; he clutched the doctor, begging him not to let "them" burn him up "down there," pointing to the corners of the room; reiterating this with many groans, sobs and cries; he frequently got out of bed and kept up the same agitation all day, but with complete orientation. The temperature was 104° on the evening of the 24th, continued about 103° the 25th and sank to 100° and normal on the 26th; for some time it reached 100° on several evenings (May 25 and 26, and June 11 and 14), and occasionally in the morning (May 7 and 16); with the sinking of the temperature a quieter condition returned. But up to July 15 he maintained an undercurrent of fear with slight exacerbation of his complaints and of interpretations of the following character: a patient gave H.'s wife two letters, which she mailed for him. H. then felt "pretty sure" that he was going to be put to death. "I heard people in the dining room saying I was going to be put to death with big hatchets and an attendant said this is the last meal you will ever eat with me" (May 16). The only more complicated interpretation was maintained from May 2 until October 21, when he recognised it as imagination, but was sure he heard the talk that time: Dr. G. brought "Judge Almy" from C. to the door of the patient's room and said—"Is this the man you sentenced?" The judge replied "Yes," and Dr. G. said "that is all I want to know." This episode is referred to about April 26. H. was then sure that it was a death sentence. June 13 he said: "I am sure of it. I can see it. I can see it everywhere. I am to be taken

to my room; five men will kick me with spurs and then take me to ward 'S' and string me up on the shower baths of scalding water." He at times maintained having heard, at other times having made out, that his attendants said: "Jesus Christ, when I get hold of him, I'll stitch those things into him"; or he heard that they were going to take him into Gage Hall, to take out his eyes, and "they are going to cut my testicles out the first thing." "Who said so?" "The attendants and all of them; they know all about it. They are going to chop me up to-night" (June 13). He often asked up to July—"Doctor, will you let me live another night?" or "for four days?" and then on the second day he said: "Doctor, did you mean when you said I should live over the fourth that I should not be tortured?" He realises that he was committed for an attempt at suicide, which was insane, but does not admit that his prognostications are ridiculous. He repudiates their being in any way as insane as those of B., who maintains that he is to be buried alive. His memory and orientation are good throughout, and there is at no time any foundation for his ailments. His complaints are usually produced with a mask-like countenance.

July 16, 1899, he displayed perfect insight—"They were all imaginations and I am going to brace up;" he even wanted to work; but August 20 he is again described as groaning, and weaving back and forth, slapping his hands. "My wife and furniture are all going to be lost. The probate court sits the fifth, and I am going to lose all, and everything will be swept out and my wife will be brought up here crazy." "How do you know?" "I told myself so; I made her so; no one told me." After four days of noisy lamentation, he continued depressed and restless till the middle of September. From then till November 3 he was quiet, out of doors every day, hardly volunteering anything, except occasional requests for "sleeping powder," which he did not get, or need; he, however, answered the questions promptly, was perfectly oriented as to time and place, remembered clearly all the previous conversations, called the episodes with Judge Almy "all imagination." "It was a man who looked like him, and I heard the talk about the sentence." He did not really hear the attendants say that he was to be killed; but when he heard them talking indistinctly he imagined it was about his death, "because I felt so bad." He was calm and rational, not especially depressed or anxious, only a little scared occasionally at night when he did not sleep. But he knew that it was foolish.

November 3, 1899, there was a sudden turn for the worse. An attack of diarrhœa with incontinence confined him to bed for seven days. Saliva was constantly flowing from his mouth; he often groaned and moaned: "my God, my wife is going to starve to death. She will be turned from her home." From November 10 he was up, but required bathing every day, very often asked for something (as for milk, water, to go to the closet, &c.). He spoke of the sheriff selling his household goods, but did not express any anxiety. His face became very oily and the nose especially became crusted with a simple seborrhœa, and roughened up to the forehead. November 29, the patient made an attempt at suicide. He asked to go to the closet, filled a bowl with water and attempted to drown himself in it. From November 30 he was kept in bed on account of weakness, diarrhœa, filthiness and destructiveness. He groaned and moaned often, had his mouth full of saliva and spoke almost unintelligibly, whereas before he never had shown the slightest speech-defect; he was restless, often trying to tear the bed-clothes; on several occasions he had a number of spasmodic twitchings of the arms, but they seemed to appear only when he thought himself watched. He took his food well and showed no other symptoms beyond the peculiar pseudo-stupor. January 3, 1900, his temperature was 99° at 8.30 a.m. Two hours later a rattling in the throat was noticed; the patient's temperature was then 103°; he was lying on his back with 44 respirations per minute, fixed eyes (but reacting pupils), considerable blind agitation and occasional trembling of arms and hands, especially during the forcible expiration; once or twice he drew his face in contortions, showing the teeth and lifting the arms during the inspiration in apparent distress. The heart beats were covered up by the tumultuous breathing. The radial pulse could not be obtained on account of the agitation, and the inguinal pulse was weak and irregular. The percussion of the lungs was negative. The patient flinched when pricked; both the knee-jerks were exaggerated. Death occurred at 11.30 p.m., January 3, 1900.

The autopsy, made three hours later, showed pulmonary embolism with beginning engorgement of the left lower lobe; several old adhesions of the left lung, a few yellow patches in the mitral valve and the beginning of the aorta. The weights were: left lung 280 grammes, right lung 340 grammes, heart 265 grammes, spleen 110 grammes, right kidney 155 grammes, left kidney (?), liver 1,475 grammes. The source of the embolus was searched for in vain.

The skull had almost completely obliterated sutures with the exception of the posterior end of the sagittal, a fair amount of moderately hyperæmic diploe; a moist non-adherent dura; the pia of the convexity was very slightly whitened along the blood vessels and considerably œdematous in the depending parts; the lobes separated easily; there were no granulations of the ventricles. The basal arteries were white (not sclerosed) and contained clotted blood. The weight was 1,505 grammes.

The microscopic findings were as follows:—

The sections of the *paracentral lobules* stained in methylene blue, without embedding, present a combination of acute alteration and Hoch's alteration in most cortical elements. The large pyramids have, moreover, throughout the typical axonal reaction and are little modified by the other accidental terminal alterations. The blood-vessels are not affected and the general arrangement of the tissue is normal. In the *frontal cortex* the axonal alteration is demonstrable only in the very few large pyramids. The calcarine cortex and the cerebellum are also free, and the latter not affected by the acute alteration. In the *medulla oblongata* all the cell groups showed more or less numerous instances of cells with typical axonal reaction, far in excess of what is usually found. Not only the cerebral afferent cells but even those of the nucleus ambiguus were extensively affected.

The Marchi specimens give the following findings:—The *paracentral lobules* show a large number of degenerating fibres of varying course, but mostly descending with the marrow of the convolution and appearing in or beneath the deepest layer of the cortex, and only rarely in the third layer. In the *first frontal convolution* there is only here and there an isolated degenerated fibre. The *medulla* shows scattered black lumps in the pyramidal tract, in the region of the cerebellar afferent tract, throughout the nuclei of Goll and Burdach, and among the arcuate fibres and the fillet. *But the degeneration in the pyramid is much slighter than the findings in the paracentral lobule would make one expect.*

The *cervical cord* has a marked degeneration in the postero-median columns more on one side than on the other, few lumps in the postero-lateral column, more in the lateral columns and in the direct pyramidal tract and relatively few in the anterior ground bundle.

Case 7 (from Dr. Fleming's service).—*M. D., a woman of* 33 *years, of French-Canadian origin. Melancholia with marked perplexity, leading to a peculiar delirious agitation with twitchings*

and attacks of fright during an irregular partly febrile episode of enteritis lasting nearly four weeks.

The maternal grandmother of the patient is 106 years old, and has been peculiar mentally in her old age. *The oldest child* of the patient is an epileptic imbecile. The other children are normal, also four brothers of the patient. The mother died in December, 1899, of unknown cause. The patient was born in Canada, 1867. She went to school very little, if at all, and was obliged to weave in a mill at the age of 11. She was rather weak physically (on account of the early work) but normal otherwise. At the age of 21 she married Mr. D., after two years' acquaintance; of very retiring disposition, she devoted herself to the housework and the children, and to learn to read was her only pastime. Her husband calls her an intelligent and good housekeeper, of temperate habits and cheerful, never gay and never despondent.

Two of the patient's brothers of 12 and 22 years boarded with her; two other brothers lived in the neighbourhood. In 1896 her husband became a Methodist and they began to read the gospel, and the wife and two younger brothers were also converted. The two other brothers would often come to the house and worry their sister by telling her that their grandmother, "who was a witch," had given her the imbecile boy and had changed her religion.

On December 21, 1899, the patient's mother died. From this time the patient became greatly depressed, cried often, and finally almost incessantly day and night for four or five weeks. She would say "my mother is dead and my grandmother too" (she had only heard that she was weak), and she would cry out "I am damned," because the grandmother was dead. Up to February 2 she did all her work, except three weeks' washing which she neglected. She thought she saw her mother in dreams, but she did not hear her speak, nor did she have any hallucinations when awake. During this period her brothers persisted in coming to the house during the husband's absence, and in worrying the patient about her conversion.

Anxiety began to make the patient sleepless. She would not go to bed. She heard people come in downstairs, and although she did not hear them speak she knew they wanted to kill her. "As soon as I fall asleep they will come up." Hence she sat up most of the night. During the night of February 23, 1900, her husband saw her jump from the second storey window, but succeeded in catching her arm and in pulling her in. She was very much excited, she "had married the devil, and she and her children were to be killed." In a paroxysm of anxiety she

had tried to commit suicide, and it was impossible to quiet her. She was taken to the Poor Farm the next day. She was locked up and restrained several days, wanted to go home, but became more quiet, and passed into an attitude of timid apprehensive bewilderment. She never spoke to any of the inmates, but often picked their clothing and followed them around. She would stand by the matron from five to thirty minutes without saying a word. When her relatives came she recognised them, but would only sit by them holding their hands, looking at them in a puzzled way, and would hardly say more than four or five sentences in an hour; she wanted to go home, objected to being shut up, and said that she would do better at home. She did not know clearly where she was; she was not untidy, but required dressing and undressing.

On April 4, 1900, the patient was admitted to the hospital. She was a woman of about five feet, weighing only about 87½ pounds, hair brown, rather thin and fine, eyes gray, complexion sallow. There were several ecchymoses on the left leg and arm and around the left eye. The palate shallow. The lobules small and adherent; the right lobe of the thyroid somewhat enlarged. The recti abdominis muscles were separated, and there were numerous lineæ albicanthes. No disorders of the senses were discovered; the knee-jerks were brisk, the wrist and elbow reflexes present, the plantar reflex normal; ankle clonus and abdominal reflexes were absent. There was a slight tremor of hands on rotation and a slight gelatinous tremor of the tongue. No other motor disorders. The internal organs showed nothing abnormal. The urine was reddish-yellow, somewhat clouded, acid, of specific gravity 1030, free from albumen or sugar; no casts.

On April 5 the patient was found sitting on a settee. When spoken to she looked with an expectant half-puzzled expression without fear. To repeated questions she said she was fairly well, and spelled her name; she was difficult to understand, as she spoke hesitantly and hardly above a whisper, and in very poor French. She was led into a room and she sat down with a simple inquiring expression, slightly parted lips and sleepy eyes. While this description was dictated she remarked: "They brought me in a carriage." When smiled at she smiled. "Where do you live?" "I live here." "What is this?' "I don't know; I don't know all the rooms. Doctor, have I got to go into all the rooms here?" "What kind of a house is this?" "It is here." "What do you call it?" After it was

explained she was asked what had been said. "You said the hospital." "Where?" "In Worcester." "Are you ill?" She hesitates, looks around uncertainly, and finally says after several starts: "I am not ill." "Is your head troubled?" After long hesitation and numerous repetitions of the question, she whispered "no" to the side with a slightly embarrassed negative expression. "What did I say this was?" After many repetitions of the question, she said, "The hospital of St. John, you said." When the physician left her, she also left her chair in a simple, contented, though uncertain, hesitant manner. The general picture was that of a poorly-nourished woman, who is puzzled, uncertain, not able to grasp the situation for any length of time, but evidently anxious to do something. She follows the attendants with a perplexed look. The emotional tone is rather indifferent.

April 12, she presented very much the same picture: she is hesitant, perplexed, looks around puzzled, but remembers the writer. "Yes, I think I have seen you before." "Where?" "I was in a room with the mistress of the house (pointing to Dr. F.). Is she the mistress of the house?" "What house is this?" "I don't know, they took me here." "What did I tell you it was?" "You told me" (here she paused and began to cry a little) "You told me—they took my ring." "Who?" "A woman that brought me here. They brought me here to make me live. I would like to have my shawl," pointing to her shoulders. "In what city are we?" She looks around studying, breathing deeply, and after many questions she mumbled something unintelligible. "Are you in Canada?" She brightens up a little and finally says, "Yes." "Can we do anything for you?" Here she begins to whimper and finally mumbles something. "Do you need anything?" "Make me live." "Are you ill?" No answer. "Are you dead?" She smiles and says: "I don't want to die either." "What do you want?" "I want to stay with those people (*créatures*) out there, with the people" (*monde*). "Are you afraid?" "If one does not do anything, I'm not afraid, but if one does something, I am afraid." "Why are you so sad?" She begins to breathe more rapidly, whimpers a little and says, "I want to—I want to—" Then she pulls out her handkerchief and blows her nose on the extreme corner. "Do you need anything?" "No." The *perplexity* and *hesitation* and *slight uneasiness* are plainly in evidence. The answers were difficult to elicit, but when the patient once spoke, she did so without retardation.

April 19 and 26, the picture was similar. In the meantime she ate fairly well, but lost flesh; she weighed but 78½ pounds. On April 30, she had to be helped in undressing and resisted somewhat; usually she sat quietly on a settee with an expectant half anxious face, and occasionally worry, or she followed the attendants, or was anxious to have them sit down beside her. At times she suddenly jumped up, shook her dress and assumed a frighted expression. On the 19th she was greatly perplexed and uneasy; sitting on the chair with the physicians, she leaned over to the left side while slowly peering around, then made symmetrical movements with both hands around her waist, and then down the thighs and legs. "What do you want to say?" "I am all right" (without changing her puzzled attitude). "Please go and shut the door." She turns around slowly, still leaning to the left, turning her head to the right to the door. When requested again, she gets up slowly and awkwardly, and remains standing near the door; after about a minute she puts her hand on the door knob and draws the door open; then turns puzzled to the physician, wipes her face and takes hold of the door again. "Come and sit down!" She opens the door again. "Now close the door and come and sit down!" She leaves the door open and comes sideways with slow steps and sits down. "You know this house?" "Yes," but nothing could be elicited. "Have you pains?" "Why do you look so?" Her eyes became a little flushed and the face more troubled. "Can I do anything for you?" She begins to breathe more hastily and looks as if she would begin to cry. "Well, what have you to say?" She looks puzzled, the head slightly stretched forward, makes a few starts as if to speak, but remains silent. On April 26, she had the same expression of extreme uncertainty, reached out her hands in an uncertain half ataxic way and held the hand; she said: "This is the hospital." "And what is it called?" "My name is Mathilde D." Beyond this nothing could be obtained.

On May 10 she appeared equally hesitant and perplexed. "Do you feel sick?" "I am—I am—I am—I am not sick; I don't want any medicine. They make me take medicine and it does not do me any good" (making motions with the hand over the stomach).

On May 17, she had been in bed three days with a cold. "How are you?" "I have a cold" (rhume). After this frank response she shut her eyes, would not answer, and resisted slightly.

On May 11 her temperature was 99·8°, from May 12-13 101° to 101·6°, from May 14-22 99° in the morning and 100° in the evening. The appetite was poor; food was taken on persuasion only, and the sleep began to sink from eight hours to almost nothing on the 23rd and 24th, when the temperature rose above 102° and in the evening to 104·6°, to sink to the normal again May 25-29. May 20: a blood count was made showing 4,000,000 red and 6,500 white corpuscles. Diarrhœa came on; in one of the five stools of May 22 there was some blood. The abdomen became slightly distended and occasionally a little painful to touch. May 23: the blood count was the same (4,000,000 and 6,500), with a temperature of 103. The diarrhœa was arrested until May 28, and later reappeared irregularly. Widal's test was negative (May 25).

The *mental condition* became that of a *perplexed agitation* with occasional attacks of fear and screaming. May 23, when her *husband visited her*, she first cried "eh—eh—eh," &c., stretched arm and hand against him and turned the head away, but then at once took his hand and kissed him. She knew him but asked no questions and answered none except "Are you in L. or in W.?" "In this little corner." "Do you worry about the children?" "No." She often said "T." (the name of the brother who had troubled her), and at times cried and screamed, and tossed the arms around. She called the husband back but had nothing to say. May 24: she was found with dry, cracked lips, sordes, but moist tongue, with a number of aphthæ, somewhat distended, tympanitic and gurgling *abdomen*, hardly sensitive to palpation; the skin of the chest and arms red and scratched from the restless friction and picking. She makes uncertain, unsteady movements with the hands, mostly symmetrical, often with one finger down on the body or lips, moving along tremolo-fashion or making a poorly-defined picking movement. The facial expression varies; a smile she answers with a sardonic smile. She pays prompt attention but *from time to time she gets the expression of fright*, with increased restless, partly defensive, movements of the arms, and a stare and scream. "Yes." "What do you want?" "I want Caspar." "Do you want Caspar?" "Yes." "Your brother?" "Yes." "Do you need anything?" "Oui,"—long drawn out, while she puts her fingers to the lips, and ending in a scream and a movement of the arms to cover the face. "What do you need?" "I need—I need nothing," again in a calmer, half-whispering tone. When asked to show the tongue she said "oui," answered a smile, then

began to stare, and screamed and struck at the physician, looked away crying "docteur, docteur," and was quiet again. After the tongue and throat were examined, under slight resistance, she was asked, "Are you afraid?" "Yes, oui, oui," and again she screamed, staring with a slight *nystagmus* and ending up with a "non-non-non-non-non," returning to her palpation movements. "Do you know me?" "Yes." "Who am I?" She looks and finally says "no," and "I am afraid of you," with a new attack of fear which was rapidly pacified. Later she again answered a smile. She often repeats a few words of a question or answers "oui" without always grasping the sense. To a repeated "Who came to see you yesterday?" she said "Nobody." "Your husband?" "Yes." "Are you sad?" "Yes." "Why?" "I don't know," just with the lips. Then after a long pause, with movements of the hands and lips and another screaming attack, she turned to Dr. F., saying, "bonjour, bonjour." No orientation; she does not shake hands when the physician leaves her.

From May 26, she was more quiet, slept more, and took food and medicines better; she still had from one to five movements most days. The pulse remained about 110. On May 31, although the temperature had again risen above 100° and 101°, she spoke rather more freely and was less frightened. "Good morning; how are you?" "Good morning; I am glad to see you." "Do you want to take me away?" "I have 2,000 piastres at home; my father is dead, but he left me 2,000 piastres. Won't you take me up to X. (unintelligible)? I want to get out of here." "What is this?" "A hospital." "Where?" She looks to the ceiling and answers: "I am going to give you money if you take me away." To many questions whether she is sick, she answers nothing. She shows the deep red, slightly parched tongue, but gets it hardly beyond the teeth. "Do you need anything?" "Yes, I want to eat." "What city are we in?" (repeated)—no response, nor to the question whether the husband had been here, who the physicians were, whether palpation of the abdomen hurt. She mumbled several times something like "I want to go away." "Have you pain?" "No." No speech disorder was established at any time.

The first week of June she grew more sleepless; she ate poorly, cried often, and from June 6th she began to show a peculiar twitching of arms and body, with occasional twists of the head; she picked herself almost constantly, and at times straightened out suddenly and screamed at the top of her voice,

as if in pain. She is resistive to almost all the attentions of the attendants and quite untidy. She took her food only on persuasion, from the spoon.

On June 8 she was found in great *perplexed and helpless agitation*, very much emaciated, with a facial expression reminding one of a miniature of Rameses' mummy, the angles of the mouth pulled down, the profile almost cutting, the forehead occasionally in a play of contractions in efforts to open the usually but half-open eyes, accompanied by even further downward movements of the angle of the mouth, or in vain efforts to say something with pointed lips; the eyes and mouth are red and slightly excoriated from the continual palpation with the fingers and the rubbing; the olecranon region and the wrists and the chest are scaly and almost copper-coloured from the friction in the continual movements of the hands, which go on fumbling tremulously, often with lifted elbows, digging with the index on the bedding or the face or the chest, or pulling the sheets a little. The legs are pulled up and held rather firmly, but the resistance is overcome with some effort. The plantar reflex is prompt on both sides; on the right the toes go down a little and then slightly up, and on the left they all go downward. The knee-jerks are somewhat exaggerated; no ankle clonus is elicited. The patient had been too weak to get out of bed for over a week; twice she had been seen to break down suddenly when she had walked into the hall.

The patient appears to notice the physicians and that they speak to her, since she looks up at once when spoken to. In her fumbling and grimacing she makes vain efforts to speak. "Show me the tongue." She opens the mouth and puts her finger in tremulously. Another time the request caused her to open the mouth and show the lips; but she does not protrude the tongue. She takes hold of a watch held out to her, in a very tremulous and awkward way, and mumbles "Trois heures" and "mo-mo-mo." It was 9.12 a.m.

The examination of the internal organs is very difficult owing to the unrest, the irregular and at times tumultuous breathing, and the frequent and sudden contractions of the pectoral and clavicular muscles. No dulness or râles are discovered. The respiration varies from 20 to 25 per minute when the patient is calm, the pulse between 100 and 110, the temperature 101·8°; the sleep is much reduced; food is often refused. The *diarrhœa* was not constant, as she would several times go two days without a stool, though at other times she would have from two to four

stools a day, generally during the afternoon, from 3 to 8 p.m. The tympanitic condition of the abdomen has disappeared. On June 8 she began to refuse the food, pointing to the throat, making a motion as if it hurt her or as if she could not swallow. She took the food into her mouth, but would be unable to swallow it. She shook the head when more was offered her. The sleep became more impaired, and the general condition weaker.

On June 9, at 5 p.m., the patient showed signs of collapse, the fever had risen to 104°. She stared intently at the ceiling, reached up her hands as if attempting to grasp something, mumbling continually. Death occurred at 11.45 p.m.

The treatment had been mainly palliative (against the diarrhœa) and trying to effect rest and better nutrition. The diagnosis first was anxious melancholia, although the depression gradually gave way to a more or less anxious perplexity. The diagnosis of a "central neuritis" became very probable when a typhoid infection could be excluded, and the agitated weakness gave signs of jactation. The *autopsy* was made twelve hours after death by Dr. Dunlap.

The body is that of a small *emaciated* woman. Rigor mortis complete. Hypostasis marked over posterior part of thorax and buttocks. Panniculus adiposus 1 mm., pale yellow. Intestines but moderately injected, filled with gas, moist and free. Omentum retracted, containing very little fat. Appendix normal. Liver not visible anywhere below the rib margin. In the pelvis about 10 cc. clear fluid. Diaphragm at the fourth interspace on both sides. The lungs retract considerably. No pleuritic adhesions.

Pericardium free, with 8 cc. of clear fluid. The left heart fairly firm, the right lax. The pulmonary artery contains a soft mixed clot.

Heart, 135 grammes.—The right auricle contains a small amount of non-adherent mixed clot. The right ventricle is pale, the walls from $1\frac{1}{2}$ to 3 mm. The tricuspid valves are thin and normal, also the pulmonary valves. The left auricle is free. The mitral ring admits two fingers easily. The left ventricle walls are from 8 to 13 mm. thick, pale, brownish-red; the valve curtains are normal.

Left lung, 265 grammes.—The upper lobe gray with a purplish tint. The lower lobe a little firmer and a darker purple. A few scattered pin-head sized *sub-pleural ecchymoses*. The consistency is soft throughout, a little boggy in the dependent parts.

Section: Grayish-pink in the upper, red in the lower lobe. The cut surface of both lobes is smooth and from each a *bloody fluid* and *some froth* can be expressed; much more abundantly in the lower lobe. The bronchi contain frothy pink mucus. The larger vessels are free, except for a few soft dark clots.

Right lung, 300 grammes.—The middle lobe and anterior part of the upper lobe gray and soft. Lower lobe and posterior half of the upper, purplish pink with projecting light pink lobules and on the outer surface of the *lower lobe, three dark red, almost black*, elevated resistant *nodules* about 8 by 8 mm.

Section: Upper and middle lobes in gray portion normal and dry. Lower lobe and posterior part of upper, red with smooth surface; considerable *frothy pink fluid* flows from the upper lobe, practically none from the lower, from which, however, a bloody fluid with fine froth can be expressed. Section through the dark areas shows the nodules to extend about 8 mm. below the surface.

Spleen, 110 grammes.—Flabby, with considerable pulp. Trabeculæ very indistinct. Substance on section quite friable.

Left kidney, 95 grammes.—Capsule splits considerably without tearing the cortex. Surface pale and smooth. One *yellow speck* 1 mm. in diameter. Cortex 3 to 4 mm., pale, with yellowish streaks. The glomeruli appear as translucent points.

Right kidney, 115 grammes.—The capsule tears the substance considerably. External appearance, section and glomeruli similar to left kidney.

Liver, 1,080 grammes.—Smooth, reddish-purple. Section: light purple, with very indistinct lobules. One gall stone the size of a large cherry.

Intestines.—About 13 cm. from the ileo-cæcal valve, in the ileum, is found a shallow ulcer with rather indefinite edges and slightly elevated peripheral zone (about 3 mm. in width). Part of its base has a translucent, healed appearance. The whole depressed area is 16 by 4 mm., the deeper area with ragged base 8 by 1½ mm. No enlargement of Peyer's patches; the mesenteric glands average about 6 mm. in diameter.

Skull, 373 grammes.—Symmetrical, smooth; sutures indistinct. Sulci shallow. Considerable diploe throughout. Slight pacchionian depressions along the sagittal suture.

Brain, 1,100 grammes.—Rather flabby. Lobes separate easily. Pia of convexity thin with but little fluid in the sulci. Cysterna clear. Vessels of base thin. No granulations of the ventricles.

Anatomical Summary.

Emaciation. Small erosive ulcer in ileum. Ecchymoses in one and slight œdema in both lungs. Dark nodules (and beginning pneumonia?) in the right lower lobe. Gall stone.

Microscopically the lower lobe of the left lung contains intra-alveolar clumps of streptococci with barely beginning reaction in the tissue. A small dark nodule shows hæmorrhagic and fibrinous infiltration of the alveoli with but few traces of cocci.

The heart muscle shows beginning fragmentation. Liver and kidney had no characteristic lesion. The intestinal erosion shows hardly any granulations.

When the *axonal reaction of all the Betz-cells* was established, the brain was subjected to an extensive study with the *osmic acid reaction*. The *right hemisphere* yielded:

(1) Sections through the radiations of the frontal lobe as far back as the foot of the central fissure (fig. 3, *a*, and fig. 4, *a*).

(2) Sections through the radiations of the upper part of the motor area and the upper parietal lobule (fig. 3, *b*, and fig. 4, *b*).

(3) Sections through the auditory radication (fig. 3, *c*).

(4) Sections transversely through the hemisphere, at the level of *d* (fig. 3 and fig. 4).

(5) Transverse sections through the occipital lobe.

The *left hemisphere* was trimmed for a series of transverse sections of the basal ganglia, and the somewhat defective results of the study of the right occipital lobe called for a study of a series of sections through the left occipital lobe. The rest of the *brain-stem* was examined in a section through the mid-brain, and several transverse sections through the hind-brain, in the trigeminal segment, the pneumogastric-hypoglossal segment, and the first cervical segment.

The result is recorded in the drawings, and is here summed up as follows:—

(I.) The segmental elements, as far as examined, were little affected. The motor cells of the hypoglossal, the spinal-accessory and vagus, and the facial nuclei, show varying degrees of irregular rarefication of the stainable portions and in each section from two to four cells, with typical axonal alteration in each nucleus. The same holds for the first cervical segment. Only few root-fibres are affected. Among the cells of the nucleus reticularis, in the auditory-facial-abducens segment, there are many with axonal alteration, but a small number perfectly normal. Many cells of the lateral nucleus have the axonal change. The

respiration-bundle has but few large lumps of decaying myelin, and the caudal root of the fifth nerve is practically free. In the Gasserian ganglia the cells show moderate shrinkage or none. They vary in diameter between 25 and 60 μ. The free marginal border is practically absent. The preponderating cell-type is

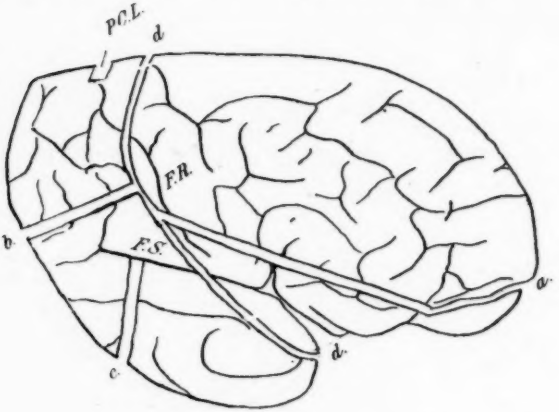

FIG. 3.

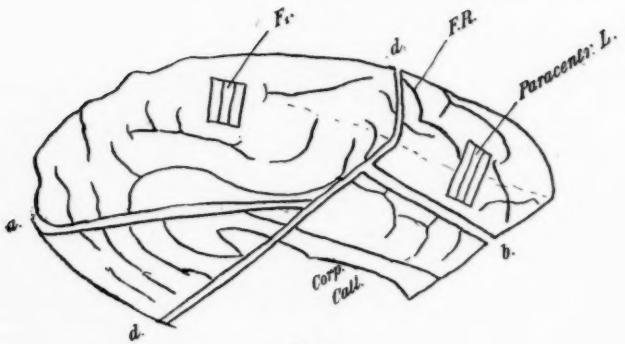

FIG. 4.

that with a marginal zone of rather large stainable lumps, gradually decreasing in number and size until a free perinuclear zone is reached; in a relatively large number of cells the stainable portions are practically limited to the marginal ring, and in about an equal number the medium-sized lumps are rather evenly

distributed from the periphery to the nucleus. The nucleus is normally fairly central and only in very few cells broken through the marginal ring of stainable portions, so that an undoubted *axonal reaction* could be established. It has usually a plain membrane, a diameter of 11-15 μ, a nucleolus of 3·5-5 μ with frequently plain apposition bodies, whereas the sap and lininframe remain free of other blue granules in the Unna specimens. There are but few "fatigue-nuclei." Moderate pigmentation, usually perinuclear. The sympathetic ganglia present cells of the most varying types, from the rested cell, with even distribution of the stainable substance and clear central nucleus, to those with extensive dissolution of the stainable substance, and eccentricity of the frequently darkened nucleus, even beyond the marginal ring of granules.

(II.) The fore-brain and its dependent parts :—

(1) The *fillet* is slightly affected from the nuclei of Goll and Burdach (many of whose cells show the axonal reaction) to the ventral nucleus of the thalamus, and to the posterior corpora quadrigemina. The *optic tracts* are not affected.

(2) The *thalamo-cortical connections*, as seen in sections through the basal ganglia, are intensely involved to the following extent :—

(*a*) The radiations *between the lateral nucleus of the thalamus and the motor cortex* are very deeply blackened (see fig. 9).

(*b*) The *optic radiation appears* to be free in the external geniculate bodies.

(*c*) The *auditory radiations* have a small number of degenerating fibres.

(3) The *medulla of the convolutions and the centrum ovale*:

(*a*) The *motor region* and its projection fibres are profoundly affected. A few of the blackened fibres penetrate the polymorphous layer of the cortex. The presence of degenerating fibres is not absolutely limited to the central convolutions but overwhelmingly so ; backward the degenerating fibres are sharply limited to the posterior central gyrus. Degenerating fibres to the corpus callosum are not very numerous. From the irregular course of some of the fibres one might well infer that also association-fibres are involved.

(*b*) The white matter underneath *the first temporal gyrus* and the transverse temporal gyrus alone have degenerating fibres, especially the former, while the second temporal gyrus is devoid of them (see fig. 7).

(*c*) In the *occipital lobe* the maximum degeneration involves

the forceps, and it is easy to follow into the splenium of the corpus callosum on the one hand and into the cortex on the other, especially into the lateral cortex (see figs. 15 and 16). The optic radiation is relatively free, and also the inferior longitudinal fasciculus. Whether the degenerating fibres of the subcortical layer of the calcarine cortex belong to the forceps, is not definitely settled.

(4) The *cerebral efferent elements* have suffered to the following extent :

(*a*) The *pyramidal tract* shows a marked decay of myelin-sheaths of some of its fibres. The relative number is smaller than the appearance of the cortex would make one suspect, though the difference is not as marked as in case 6. This discrepancy led to a study of the thalamus which shows that so many of the degenerating fibres were thalamo-cortical.

(*b*) The cerebro-pontile fibres are only very slightly involved, so that Türck's bundle as well as the median part of the crus appear free.

(*c*) The fornix is free, with the exception of one or two fibres in a section.

(*d*) The corpus callosum has fairly numerous degenerating fibres in the splenium and about the region belonging to the motor area, but not very clearly limited.

The *typical axonal alteration* was found to be present in the paracentral lobule in all the Betz-cells and in many of the large pyramids. In the frontal lobe the alteration was much rarer throughout the cerebral cortex, the cells are very pale, with great reduction of the stainable substance but practically no diffuse stain of the non-stainable parts. The *blood-vessels* are nowhere infiltrated or thickened, but slightly distended.

III. The *cerebellar apparatus* is involved in three points :

(1) There is a distinct degeneration of a number of fibres in the restiform body in its course around the dentate nucleus to the vermis.

(2) Many of the cells of the dentate nucleus show a beginning of axonal alteration.

(3) The cells of the roof-nucleus have pronounced axonal alteration. The Purkinje cells are free.

Case 8 (from Dr. Budd's service).—*H. B. P., aged* 56. *Alcoholic senile deterioration with stuporous delirious episode. Collapse.*

The father of the patient had a shock at 68 and was mentally feeble afterwards, and died of a second shock. The two brothers and three sisters are free of hereditary features.

The patient was born in Maine, 1844. He went to the war at 17 and left the service in a year with "black measles and dysentery." "He did not recover his health for many years." He worked on the farm two or three years, and then became lineman on telegraph-lines, a good worker with highest wages. In this work he sustained *several injuries* from falls; he also was given to alcoholics and venereal excesses (many drinks of raw whisky every day; no venereal infection known of). He married and had children; about 1890 he left wife and family on account of his drinking and abusive and hot-headed disposition, and since then he has boarded in different places.

November 10, 1898, he was run over by an express-waggon. He was admitted in the Boston City Hospital with lacerated wounds of the face, and the next day he developed a probably *alcoholic delirium* which lasted nearly a week and necessitated the use of a straight-jacket. He was discharged well November 26, but his daughter thinks that he began to fail after this. He drank more, "seemed to stay half full all the time," and it happened that when he took down a wire for repair he went away forgetting to put it up again. He had to be given the easiest work, and about February he had to give up even that, because he was unable to go to the shop on account of physical weakness, especially in his legs. He spoke of getting a pension from the government and was very impatient about it.

For the last month he wandered about the house acting like a child, but up to within a week before his commitment he talked rationally and sensibly and was neither troublesome nor violent; his memory only was very defective.

About a week before his admission to the hospital he began to produce a confused talk about Heaven; that his son was being "buncoed" out of Heaven, that there was a war going on in Heaven, &c. He began to go two to three days without eating, knew those about him, but was very restless. He would be sitting down talking and suddenly jump up and go out into some other room, look around and come back again, saying, "It's all right," then he would sit down, only to get up again in a few minutes. He slept very little at night.

No evidence of delusions of persecution, of reference, of robbery or of expansive ideas, or of hallucinations could be obtained from informants.

Lately he has been very weak but not in bed. The last day at home he was arrested at the instigation of the boarding-house keeper, because they said he grabbed one of the children by the

throat. The daughter says he was not cross and irritable, and thinks he really intended no violence. He also threatened and tried to jump out of the second storey window.

On admission, May 19, 1900, the patient required assistance to get into the ward on account of his apparent weakness. On the evening of the 19th and the morning of the 20th, he had no idea of the city or nature of the place, but refused to assent to absurdities (stable, &c.). He twice said he came from Omaha, Nebraska. He did not know the year, month or day, and when pressed for the season, said it was winter. He was told the place, but on the evening round of the 20th he did not remember being told, and he had no better orientation, except that he said that the doctor "acted like a doctor," because he felt his pulse. He did not remember seeing him before in the morning, but said he had seen him before he came here. He constantly said there was nothing the matter with his mind. On admission his pulse was very irregular, intermittent, dropping one every two or three beats.

Physical Examination (*May* 22).

The patient is found to be a tall, moderately slender, well developed and well nourished man with thin gray hair and beard, abundant growth of hair over the thorax and abdomen, and dark, tanned complexion. Skin somewhat flabby, warm, moist and free from eruptions. Recent abrasion on lower third of right shin, 2 by 4 inches. Over left malar-bone, a scar 2 cm. in length. No other abrasions or noticeable scars. The left *clavicle* at junction of middle and outer thirds has an old callus, the inner end overriding the outer; union is complete. *Skull* symmetrical; *adherent lobules;* palate of medium height and width; *teeth* sound but well worn. Hands well formed. Large toe of each foot is much abducted, and the *metatarso-phalangeal joint is very prominent.* Penis and testicles normal; no scar is found.

General condition.

He remains in bed, but now and then gets up and goes to the closet; does not complain unless asked, then saying nothing further than that he "is sick" (ill). His tongue is clean; appetite only fair; bowels regular; sleep only by aid of hypnotic.

Eyes.—Dark blue, well opened and prominent. Conjunctiva clear: *pupils slightly displaced toward inner side, but*

equal, 2½ mm., react to light and accommodation. Movements unrestricted. Vision normal. No hemianopsia.

Smell unaffected, and *taste* unaffected.

Hearing.—Good on either side.

Cutaneous sensibilities.—Touch and localisation, prompt and accurate. Pain and temperature tests are unreliable because of resistiveness of patient; believed to be normal.

Motor functions.—Muscular power of arms, equal but poor. Power of legs equal and good. *Gait, slow and feeble. After standing a few minutes he begins to reel as if dizzy, and complains of weakness, and he is helped to bed. Very slight individual tremor of fingers, becoming more marked on rotation. Tongue small and median, with jelly-like tremor at sides. Marked swaying in Romberg position.* No speech defect.

Reflexes.—On plantar stimulation the smaller toes flex somewhat, the large toe remains still. No ankle clonus. Achilles reflex, equal and moderate. *Knee-jerks both exaggerated, perhaps a little more so on the right. Elbow and wrist-jerks are equal and somewhat exaggerated. Cremasterics slow and long continued.* Epigastric not obtained.

Lungs.—Expansion equal and good. On percussion at the right apex, in front and over the entire surface of the lower lobe behind as far as the angle of the scapula, there is a higher pitched resonance. The respiratory murmur is relatively somewhat harsher over this area, and the expiration slightly prolonged. No râles are heard; no increase of fremitus.

Heart.—*Pulse* 64, *intermittent and irregular*, but full and of good quality. *Radials thickened, brachials slightly thickened.* Temporals not visible. Apex beat not visible, but felt weakly in the fifth interspace, inside of nipple line. *Heart boundaries.*—12 cm. to the left of median line at the sixth rib, 4 cm. to right of median line; whole width 16 cm. There is a distinct systolic murmur, rather rough in quality, best heard over the sternum, opposite the attachment of the third ribs, and better over the aorta than over the pulmonary ostium. No marked accentuation of the second sounds.

Abdomen.—Stomach even with costal margin. Liver from the sixth rib to the costal margin. Spleen not palpable. Inguinal glands felt plainly.

Urine.—May 21. Dark amber colour, clear with light cloudy sediment. Acid 1030, no albumen or sugar. Calcium oxalate, amorphous urates, epithelial cells, no casts.

May 23. Asked where his home is, he hesitates a long time,

and finally says: "Have none." "Where are your people?" "Have none." "Are you married?" "No." "Where did you live before you came here?" "Don't know."

Mental Status. (*May* 29).

Attitude and manner: Confined to his bed, he tries to run out or to pull clock down; At times he is irritable; feeds himself and is cleanly; often calls out.

Too weak to stand more than a few minutes at a time, he had been in bed since admission. At first he seemed to be ill at ease in bed, and often tried to get out, a number of times succeeding. He would then run to the door and try the handle, but on finding it locked, he would turn away saying nothing. Once he ran and reached and tried to pull the clock down from the wall and was prevented just in time. He would give no rational answer to questions about his conduct. He usually lies flat on his back. He often called out when lying in bed, saying such words as "whoa," "Over in the ten-acre lot," "Her name is Bailey," "All right," "It looks like it," "What?" "There, there," "Yes, there, east corner, 40 rods—timber land," &c., and then relapses into silence for some time. He shows but little interest in his surroundings, and answers questions in a low tone. During the first few days he showed considerable irritability when restrained from getting out of bed, but after a few days he did not try to get up so often and only showed occasional temper. Even when angry he said very little. He has been uniformly clean, always asking for assistance to go to the closet, or even to expectorate, and he has fed himself without trouble. On occasions he will absolutely refuse to eat one or two meals, and then again will drink milk when he does not take solid food. Once in a while he pulls and picks at the bed-clothes a little.

The following gives an idea of his stream of thought under a minimum of urging:—

Conversation: Volunteers rambling remarks, but finishes sentences.—"I've heard nothing from the general report. I think it is proper and right. I haven't been to the meetings only once." "Eh!" "Yes, that's all." "What have you got to say?" "I've been telling you about going into it. I suppose it was fixed over long time ago before I had anything to do with it." "What?" "What collects the seed?" "What do you mean?" "I don't know. I suppose you belong to the government, and know the seed of the womb. I suppose it would proper, you

ought to know." "What else?" "Why, I've got to reckon everything right through. When they are full they will take no more, that is all there is to that." "What is that?" "That's so, they give birth to young." "Who?" "A hive of bees isn't it? It is made by bees. They suck the seed from different meals and far different climes and different places." "What seed?' "The seed to manufacture the substance, its a honey." "Who spoke of honey?" "I don't know. That is the way I've always heard." "What have you been talking about?" "What have I? I don't know. I've been talking but little on account of"—(stops). "What?" "I didn't say anything."

Orientation: He calls the doctor as such; says it is 1900, *but does not know place, day or month; his answers show confusion.*—"What place is this?" "Wakefield Junction, is it?" "No." "I don't know." "What building is it?" "I couldn't give the first guess." "Why do you think it is Wakefield?" "Well, I don't know. I surmised, that's all." "What day is it?" "You have me now." Question repeated. "I don't know. I couldn't say." "What month?" "July." "Why do you think so?" "That will have to go. I guess you can put it down or not, just as you choose. They don't interest me very much now." "What year is it?" "Well, some say it is one, some another. It is supposed to be 1900 by some." "What do you say?" "Well, I don't know." "Is this your home?" "I wasn't born here, no." Question repeated. "Well, I come here before I was married. I had children before I enlisted." "Do you know me?" "Doctor, yes. Dr. Jones," (rising inflection). "Are you sure?" "Well, I heard you called that a good many times. I don't know as I was ever introduced to you, but I know you attended me, that's all." "When?" "Well, ever since they transferred the old Grand Army out, and transferred the old pensions on to this list to be transferred right on." "What was the trouble?" "Well, disability. I've been broken down pretty bad. You've been with me and ought to know pretty well."

Memory for remote events: generally fair, specifically poor.—"Where were you born?" "New Gloucester, Maine." "When?" "About 55 years ago." "What date?" "October 16. I was born in 1844." "Did you go to school?" "I was four years old." "How long did you go?" "I didn't go but little; went summers and winters three months each." "How long?" "I had to work the farm summers, and could only go winters." "How old were you when you stopped?" "17, I think." "What did you do then?" "I worked in a cotton factory in

Lewiston." "How long?" "Pretty near a year." "What then?" "Enlisted in the army. I wasn't there very long. I was ill considerable." "In any battles?" "Yes, I was in the seven-days' battle before Richmond." "Who was your general?" "General McClellan at that time." "What regiment were you in?" "The 11th Maine." "What date were you discharged?" "I haven't just the date." "About?" "I couldn't tell." "What year?" "I don't know." "How long were you in the war?" "You can reckon that up yourself as well as I, or better." "What year was it when you quit the army?" "It was '63, I guess, I couldn't say." "When were you married?" "Tenth day of January, 18—1880." "What?" "Eh?" "When were married?" "I couldn't tell you the day and date." "When was it?" "I don't know. I knew the day and date at the time, but I don't now." "How old were you?" "I don't know. I suppose I was 17 or 19, something like that—19 I guess." "What was your wife's name?" "E. D." (Correct). "Where is she now?" "I don't know." "Is she alive?" "She was, the last I heard of her." "Where?" "M———" (Correct). "How long ago?" "Some time ago." "Ten years?" "Oh, no." "One year?" "I've seen her in a year." "Where?" "At M———" (Correct). "Any children?" "Yes, three girls and one boy." "Where?" "The girls are all married; the boy lives with the other ones. All the children live together, except the one, Edith. She is in the same place, but different, in the same town, married." "What is your business?" "I've been working at electrical work considerably, called a lineman."

Recent memory: very variable, confused answers at times; stereotyped: "I don't know."—"How long have you been here?" "I don't know." Question repeated. "I don't know as I've been here but once." Question repeated. "Not knowing, I couldn't say, otherwise, don't remember." "A month?" "I don't know." "A year?" "Couldn't say." "When did you see me last?" "I saw you when you put those—heard the remark you made to—heard it before." "When?" "You came through here and put those pamphlets on my bed to-day." "What remark did I make?" "You said: 'Oh, yes, I prefer being on this side,' and you skipped over." "What did I mean?" "I suppose you thought it was the last day and the world was coming to an end, or an earthquake or something. A lot of foolish stuff. It sounded foolish." "Where did you come from?" "I don't know." "Why did they bring you?" "Who brought me?" "Don't you know?" "No, I don't

know, I'm sure." "Where did you work last?" "For Webb, I believe." "Where?" "I suppose it was here." "Where?" "Wakefield Junction, is it? I don't known where."

Grasp of surroundings exceedingly poor.—"What kind of a place is this?" "An armory, I suppose." "Why are you in bed?" "You said I was sick." "Are you?" "I ain't very well." "What is the trouble?" "I told you I was all broken up, and you said here was a tender spot, and you ought to know, I suppose. If you call me a healthy man, let it go at that, and I won't say anything." "Do you know anyone here?" "No." "Did you ever see them before?" "What, the people that come here to view the——? no, I don't know." "What time is it?" "I don't know" (looks out of the window), "I don't know. I should say it was considerably after noon." "What time do you have supper?" "Well, different people have supper at different times." "What time do you?" "Sometimes one and sometimes another; sometimes I don't have any." "Ordinarily what time?" "Well, split the difference between that and—and you will get it as near as I can tell you. Ordinarily I get what I want to eat.", "What time do you see me in the morning?" "Eight o'clock, is it?" "How many times have you seen me to-day?" "Three times, I guess." (Right.) Asked what the attendant did, he said: "As regards the collection of the seed, he is in it all right." Reads a watch correctly. "What am I writing?" "I think you are writing a full set of—I don't know—to have the thing progress, I guess." "What purpose?" "They have it to drill with, I suppose."

General information: (*Answers not reliable*).—"Who is president?" "What day?" "Now." "When now?" "1900?" "Do you know?" "No." "Who was shot during the war?" "I don't know." "What president got shot?" "I know a man who got shot, but no president."

Calculation: poor for combinations.—3 + 9? "27." 4 + 4½? "12." 9 + 9? "81." 13 and 7? "19." 2 + 13? "26." 9 + 12? "108." 3 + 13? "I don't know." 17—9? Pause. "*It would be* 9."

Counting.—He counts from 1 to 20 in five seconds, keeping on up to 50 rapidly.

Hallucinations: are not elicited, although he often calls out (*see above*).—There has never been any direct evidence of hallucinations, and nothing of that character can be elicited by questioning the patient.

Delusions: nothing definite elicited.—"Did you have trouble

with your wife?" "Yes." "What?" "A little domestic trouble." "What was it?" "Just a little." "What?" "Her daughter had a kid to-day." "Is that the trouble?" "Yes." "Have you any property?" "No." "Do they treat you well here?" "I've been treated all right." He occasionally jumps out of bed and tries the outside door, but never gives a rational explanation.

Insight: perhaps partial; answers contradictory and lacking animation.—"Is there anything the trouble?" "I have heart trouble bad." "For how long?" "Five or six years. Had it pretty much all my life." "Has your mind ever been affected?" "Some, not much." "What caused it?" "Liquor caused it, I presume." "How did it affect you?" "When I didn't have any it affected me." "How?" "Because I wanted it." "Did it affect your mind?" "No, it didn't affect my mind any." "Is it affected now?" "It isn't any better than it ought to be." "Have you ever been crazy?" "No." "Were you ever out of your head?" "Yes." "When?" "I was up to the City Hospital awhile." "What was the trouble?" "I got injured by a team." "Are you as sound in your mind as ever?" "You are the best judge of that." "Are you confused?" "Yes." "When?" "Well, most of the time" (in a lifeless manner). "This is an insane hospital, why were you brought here?" "Because of that arm." "Have you ever been insane?" "I told you no."

Reaction to questions: monotonous, forgetful of questions, occasionally irritable; some surprisingly accurate answers.—He lies in bed, looking out in a calm, deliberate, rather sleepy way, occasionally glancing at the speaker with a little gleam of animation, answering the questions in a low, monotonous tone, but when asked to repeat always repeating the same word or phrase, and finishing his sentences. Sometimes he makes an irrelevant or confused answer. This is more noticeable where he is persuaded to talk voluntarily (see conversation). When direct questions are put, he often makes a surprisingly accurate reply, but will perhaps make an equally foolish answer to the next question. Now and then when a question has been repeated several times, he will show irritation and elevate his tone somewhat to emphasize his reply, or sometimes saying "I told you before," or "I don't know, I tell you," then readily relapsing into his easy way again, in order to keep it in his mind.

June 16. *Restless; out of bed; diarrhœa; sudden death with cyanosis.*—Patient continued to remain in bed, observing in

listless manner those about him; occasionally speaking as if to himself as already noted. He was less restless than when admitted, but would occasionally try to get out of bed, and seemed especially restless at night, so that quite often he required light restraint. He at first showed no mental change either in voluntary speech or in reaction to questions from that already recorded. About two weeks before his death he was found to have fallen from the seat in the water-closet and to have bruised his forehead. He was lying quietly on the floor, and was not unconscious or more than usually stupid. Previous to this he had been taken to the closet after he had voluntarily started, and was always able to sit alone and to walk back with assistance. After this, however, he seemed to be growing weaker. He would often try to get out of bed, and when asked if he desired to go to the closet, he would say "yes," but he was not able to walk the whole distance, and unless he was helped he would soon sink to the floor. He was thereafter carried until about one week before he died, when he was not allowed to attempt to walk. He became noticeably weaker during the last week, and slightly more restless during the day, often pulling the bed-clothes about and attempting to get up, but when prevented he would lie back and apparently sleep. On several occasions when he succeeded in getting out on the floor, he was only able to walk a short distance, and would then sink easily to the floor, apparently too weak to go farther. There was at no time any evidence of muscular twitchings or spasms, nor any drawing of the facial muscles. He gradually became less talkative and observant. When spoken to he answered in a low tone, and generally irrelevantly. When his answers were relevant, it was thought he did not understand, but that it was merely accidental. He often took no notice of questions at this time. It was observed some two or three weeks before death that he would hold saliva in his mouth for some time waiting for help from the attendant, as he would readily expectorate when given opportunity. His expression was stolid and heavy. He often spoke about "electricity" and "wires," but not in connection with anything about the wards.

About four days before death he began to have diarrhœa, and he then was so stupid that he did not appear to notice when his bed was soiled. He had to be fed by a spoon, and passed the day lying flat on his back with eyes closed, and could hardly be aroused. He would occasionally rouse up slightly, but only for a few moments. He had not attempted to get out of bed for some

time before the time of death. The condition of the reflexes did not change.

He died June 16, in the morning, about 7 o'clock. After getting out of bed and walking through the door, he sank to the floor easily, fell backward in a heap, began to gasp in a strangling way, and to roll his eyes back. He was placed on the bed, and only breathed a few times afterward. The attendant noticed that he was at first very cyanotic over the most of the body, but, shortly after, this faded away.

Autopsy, June 16, at 11 a.m. Body of a well-developed and fairly-nourished man. Body heat present. Rigor in jaw, slight in legs, little if any in arms. Hypostasis considerable in dependent parts; purplish lividities on thighs and legs, even on anterior portion. Scrotum slightly abraded on right, lower half dark purple. Panniculus, about 2 cm., rather pale yellow. Intestines moist, but slightly injected; omentum retracted. Mesenteric glands not enlarged. *In the mesentery* of the ileum, near the scrotum, are about a half-dozen *calcareous nodules*, varying from small pin-head size to that of a small bean. Some of these lie near the intestine, others at the mesenteric attachment. Appendix normal. Diaphragm, fourth rib on right, fifth interspace on left. Liver in mammary line about 4 cm. above costal margin, nowhere visible on opening the abdomen. Lungs retract considerably; at the *right apex there are two narrow bands of strong adhesions*. Elsewhere the lungs are free; very little fluid. Pericardium contains about 10 cc. clear serum; pulmonary contains soft clot; right heart flabby and full of blood, left firmly contracted.

Heart, 315 grammes. Over the anterior surface of the right ventricle is a *thin white patch* (milk-spot), 2 by 3 cm.; *coronaries are tortuous, thickened and roughened*, embedded in a small amount of fat. *Over the left ventricle are two groups of pin-point to slightly larger ecchymoses*. Right auricle contains a dark, rather soft clot mixed with lighter clot. This does not adhere to the endocardium. Appendix contains a tough clot between its trabeculæ. *Tricuspid opening admits easily five fingers* (perimeter, 14 cm.). *Right ventricle enlarged;* contains a stringy, light clot; walls from about 1 to 3 mm. Colour rather pale. The tricuspid valve curtains are thin and translucent. *Pulmonary valves are large, flabby, thin* and free; the measured diameter of the posterior valve is 4 cm. Left auricle free, except for slight stringy, non-adherent clot in the auricular appendix. Mitral ring admits two finger-tips rather easily. Left ventricle walls are rather pale

brownish, from 2·5 to 20 mm. *Mitral* thin and normal, except for a *slight yellow streak*, a little elevated and a little roughened on the posterior surface. Aortic valves leak water slowly; thin and translucent in general, with but slight thickening near the attached margin of the right posterior valve. *The ring and beginning of aorta show small streaked yellow elevations, varying* from pin-point to 3 mm. in diameter.

Left lung, 235 grammes. Dark steel-gray, soft and uniform; interlobular fissure absent in its middle third, without any evidence of adhesions. On section, tissue is grayish-pink, dry and leathery. Small amount of dark blood is squeezed from the vessels. Bronchi very slightly injected.

Right lung, 290 grammes. Externally the same as left, except for a *patch of thickened pleura* about 3 by 1 cm. at apex, and a few emphysematous-looking lobules near this patch. Middle lobe faintly indicated by a depressed line near the anterior margin, and by a notch, 2 cm. in depth, at the fissure. Section similar to left, except for the upper lobe, which is of a pale gray.

Spleen, 155 grammes. Purple; little pulp can be scraped off; trabeculæ fairly distinct, substance rather friable.

Kidneys, left, 155 grammes. Surrounded by a thick layer of fat; substance *deep purple*. The line of lobulation shows very plainly, and the capsule splits considerably and is very thin, it tears the substance slightly. Section very dark purple. Glomeruli give a granular appearance.

Right kidney, 160 grammes. Similar to left.

Liver, 1,645 grammes. Normal in external appearance. Section rather light reddish-brown. Lobules very indefinite.

Skull-cap. Fairly regular, rather long; sutures well preserved; sulci moderately deep, in part ragged; very little diploë; marked patch of depressions about 15 mm. outside of median line, corresponding to Pacchionian granulations, visible on the outside of the dura. Longitudinal sinus practically empty, with broad fields of Pacchionian granulations. *Dura moderately adherent* to skull in frontal region. *The falx has a great number of perforations*. The inside of the dura is free; Pacchionian patches correspond to the entrance of the largest veins into the sinus. *The pia of convexity is slightly hazy, very œdematous*. The blood-vessels of the base are thin and tender, except the entrance portion of each middle cerebral. The *cisterna is thin, with a few flaky thickenings*. The lobes are very easily separated. *Weight* of the *brain in toto*, 1,375 grammes. The fourth ventricle is not granular.

Anatomical Summary.

Dilatation of the right ventricle, the tricuspid, and pulmonary rings. Congestion of kidneys, spleen, and probably liver. Ecchymoses on surface of left ventricle. Œdema and slight haziness of pia of convexity; cisterna shows few flaky thickenings. Moderately adherent dura. Perforations of the falx cerebri. Arterio-sclerosis of coronaries, aortic ring and beginning aorta; slight of one mitral flap; very slight of aortic valves. Milk-spot on right ventricle. Narrow bands of adhesion and thickened pleura at right apex. Calcareous nodules in mesentery.

The *paracentral lobules* show very strikingly the pure axonal reaction of practically all Betz-cells and some of the larger pyramids without disturbance in the general appearance of the cortical tissue. Many of the large cells resemble considerably the vortex type of fig. 2. In the frontal cortex many cells show a similar alteration—rarefication of the stainable portions of the cell-body and dendrites, and occasionally a cell with fairly typical axonal alteration (fig. 2a).

The medulla has extensive axonal alteration in almost all the cell types, except the hypoglossal cells, which differ but little from the average appearance in beginning senescence. Many of the cells of the nuclei of the posterior columns, especially those of the external nucleus of Burdach and also those of the lateral nucleus, are markedly affected. In the cerebellum the roof nucleus and the dentate nucleus have the axonal alteration in practically every cell, whereas the Purkinje cells are not affected.

The Gasserian ganglia are free; the sympathetic cells show varying types. The *examination with osmic* acid was limited to the basal ganglia of the left hemisphere, the mid-brain, and the hemispheres of the cerebellum. The number of blackened and decaying fibres is small. They are limited to:

(1) The thalamo-cortical tract of the motor and visual areas.

(2) There are few fibres in the red nucleus.

(3) The pyramidal tracts are very little affected, and the same holds for the fillet.

(4) The restiform body is considerably involved.

(5) The corpus callosum has but few degenerating fibres, and these are limited mainly to the splenium and to the motor region.

To these cases *Dr. August Hoch* kindly allows me to add another one accidentally discovered by him. Dr. Hoch's case was admitted to Dr. Charles F. Folsom's service at the Boston City Hospital, November 16, 1898. The patient was a carpenter, according to his own statement 58 years old, born in

Maine, with negative family history. The history is difficult to obtain on account of disinclination of the relatives. Alcoholism is denied.

For about two years the patient has had considerable headache and has been slow in thinking. For a year he has been unable to keep his books, but continued to work until three months before admission. Since then he has had vertigo, swollen ankles, delirium at times (?), was more irritable in his home life, had some abdominal pains, and for two or three months looseness of the bowels, which "pulled him down." He finally began to fall on the street, and one day children had to bring him home. He looked 65 or 70 rather than 58, and was really ill two weeks before he went to the hospital. He would sit on a chair with a cap drawn down over his face, and would not say anything to anyone. He finally became restless, and could not be controlled any longer by his wife. He would pick his clothes, jump over the foot of the bed, and follow his wife. He would "holler murder." His diarrhœa became worse. His wife would no sooner get him cleaned up than he would be all "mussed up again." He could not control it, although he tried to go to the closet. It took him a long time to get down stairs on account of a tendency to fall backward, as he did on the street. He never lost consciousness, and always got up by himself. He did not want to be taken away where his wife could not go with him.

On admission it was noted that the pupils were normal; arcus senilis and irregularity of arterial walls were present, but a regular equal slow pulse. No other abnormalities. November 20.—"Since entrance the patient has had hallucinations; the temperature has been subnormal; the bowels much better. November 24.—The patient is delirious and making strange movements of the hands all the time; does not recognise his wife, and will not answer questions intelligently. November 28.—Marked rigidity of muscles for the past few days; no paralysis evident; he makes peculiar purposeless movements, flourishing the arms about and often rolling fingers and thumb together; continued stupor and delirium.

December 3.—The patient appears weaker each day. The mental condition continues the same; peculiar movements also continue. Flexion of head on chest is resisted; rotation is normal; pressure on the back of the neck and tapping on the head causes apparent pain, even hyperæsthesia. The abdominal walls and the muscles of both extremities are in intermittent tonic spasm and fibrillary twitching. No elbow reflex; knee-

jerks sluggish; no patellar clonus. Slight pressure of skin causes pain. Flexure of legs on thigh causes intense pain. The plantar reflexes are increased; the abdominal, cremasteric, and dartos absent. December 6.—The patient has continued about the same, gradually weakening. Tracheal râles developed this morning, and the patient died at 2.30 p.m. The temperature had ranged between 96° and 97°, and only once, December 3, reached 97°. The clinical diagnosis was "Tuberculosis, diarrhœa." The autopsy, made two hours after death, established the existence of lobar pneumonia, adhesive pleurisy, dilatation of the lymphatics of the pleura; the dura was firmly adherent along the longitudinal sinus; the pia normal; the basal vessels absolutely free of arterio-sclerosis. The brain weighed 1,470 grammes, and was firm and macroscopically normal. The appearance of the body was that of a man of 75.

The paracental lobule and first frontal convolution were examined by Dr. Hoch and Dr. Dunlap with methylin blue, Weigert's mitosis stain, Berkeley's Golgi reaction, Cox-Golgi, and Marchi. The result was the finding of the axonal reaction in the Betz-cells, the presence of a small number of degenerating subcortical fibres, and practical integrity of the tissue as a tissue, so that general paralysis was absolutely excluded.

Similar lesions have been reported, since the note on my first two cases, by Drs. Worcester, Maurice Faure and John Turner. To these are added my eight later cases, and the one of Dr. Hoch. A careful search in the literature of melancholia and other mental disorders brought further to my notice an article by Joseph Wiglesworth, "On the Pathology of Certain Cases of Melancholia Attonita or Acute Dementia, with a report of two cases" (1883). In order to convey a better idea of the material on which my conclusions are to be built, the cases are here reproduced in abstract:—

Dr. W. L. Worcester's case of Landry's paralysis (*Journal of Nervous and Mental Disease*, 1897):—

A widow of 40, during the middle of December, 1895, three years after the menopause, began to be troubled by the delusion that she was pregnant and poisoned from a dead fœtus; she consulted a number of physicians, and finally remained in bed. Later, she read of Gov. G. having died of Bright's disease, and concluded she had it too; indeed, about the middle of March her

urine was found to contain albumen and casts, not previously found. She also had numbness in the hands from that date, and on April 9 she became awkward in their use; two days later she appeared paralysed in all the extremities. April 14, there was slight paralysis of the left facial nerve (including the ocular branch); in the extremities the parts near the trunk were more paralysed than the fingers and toes. The knee-jerks and cutaneous reflexes were absent. Diminution of faradic irritability and abolition of the same in the thighs, upper arms, and pectoral muscles, were found. There was great loss of sensibility of the extremities; the head and neck were free; the patient was rather apathetic. The *post-mortem* findings were: axonal reaction of the large cells of the motor cortex; beginning axonal alteration of the motor cells of the cord; practically no degeneration of fibres, neither in the cord nor in the peripheral nerves.

Faure's cases (*Revue Neurologique*, December, 1899) probably do not fit altogether into our picture; they are mainly cases of alcoholic-cachectic polyneuritis or delirium, from a general hospital:—

(1) A woman of 42, with cachexia, cancer of the uterus, hydronephrosis, frequent convulsions, profound mental confusion and typical axonal alteration of a great proportion of the large pyramids, slight sclerosis of the vessels and a slight degree of embryonic infiltration.

(2) A woman of 38, with tuberculosis of lungs and liver; very fatty liver, and queer incoherent and childish behaviour before death. The arrangement of the elements in the cortex is normal; but a small number of the large pyramids show axonal degeneration. No other lesions elsewhere.

(3) A woman of 30, with alcoholic-tubercular polyneuritis with mental confusion (the extent of the lesion is not stated).

(4) A woman of 32, also alcoholic and tubercular, with slight polyneuritis and mental confusion (the extent of the lesion not stated).

(5) A tubercular-alcoholic man of 35, with occupation delirium and a final temperature of 41°. A few cells show axonal reaction.

(6) A woman of 50, with acute lobar pneumonia and systematised deliria. Pigmentation of the cells; in a few the axonal reaction.

Turner's cases (BRAIN, 1899) are all the more important, since they come from a clinical material similar to mine and because

they are accompanied by an excellent description of the clinical manifestations. The anatomical condition was studied by him in the cortex only; hence, probably, his conclusion, expressed by the title of the paper. The cases are:—

(1) A woman of 45, with a history of alcoholism, fits, and melancholia in the family, alcoholic herself, depressed, with violent outbreaks for eighteen minutes; delirious and extremely emaciated. Finally, parotitis and broncho-pneumonia, with jactation of limbs, exaggeration of knee-jerks, absence of plantars, coma.

(2) A widow of 52, following an influenza (in July), became irrational, with alternate periods of depression ("a lost woman") and hilarious excitement; after four months she was excited one day and quiet the next. She had no tremor, but exaggerated knee-jerks. About the eighth month of her illness she began to fall frequently; a week later she was unable to stand, became lost and silly; one day, five weeks later, when excited, she was "rushing about the ward and falling about," and after two months of this motor disorder she died with diarrhœa. The brain weighed 1,198 grammes, and was otherwise normal.

(3) A woman of 42 was admitted in 1895, with a history of eighteen years of delusional insanity (some women were plotting against her and her husband). April, 1898, she developed agitated melancholia, began to fail. In May, her knee-jerks were exaggerated; she was much agitated. In July she had occasional diarrhœa. In September she became demented, dirty, very greedy. In November she lost the use of her legs, fell often, and was restless and bedridden. In December she exhibited involuntary jerking movements of the arms and legs, and risus sardonicus, and succumbed to exhaustion. Atrophy of the internal organs.

(4) A sober woman of 37, always hysterical, was nervous and apprehensive for a year, would not leave the house, lost the use of hands and legs, shook and fell often, and was finally committed with screaming attacks, greasy skin, absence of knee-jerks and plantar reflexes, but equal pupils, and no reaction to pricking. Mostly incoherent ejaculations. Death in four days. Slight atrophy of frontal convolution; brain weight 1,137 grammes. The radial and post-tibial nerves in degeneration.

(5) A woman, born in 1830, who had a three months' attack of melancholia in 1889 and was readmitted in 1890 with deep depression and agitation, but free of motor disorders. January 24, 1899, she took to bed with diphtheria and died February 6,

1899. There was found arterio-sclerosis of the basal arteries, œdema of the pia, atrophy especially of the parietal convolutions and a brain weight of 1,283 grammes. Turner raises the question whether the axonal alteration might not be due to the asthenic type of the diphtheria.

(6) A man of 36 whose father had been insane, was taken with incoherence, violence and raving shortly before admission; was restless, resistive, incoherent, not heeding questions, filthy, and normal physically but for involuntary jerking of the limbs and finally inability to stand. Collapse five days after admission. The meninges were very opaque over the vertex, thick, not adherent; atrophy of the convolutions. Brain weight 1,412 grammes.

(7) A man of 52 whose father had been melancholic and committed suicide, was admitted in 1892 with agitated melancholia which passed into dementia; 1894, weakness of the lower limbs came on; in 1897 he became bedridden, and died in 1898 of phthisis. There was atrophy of the frontal convolutions and dilatation of the lateral ventricles. The brain weighed 1,296 grammes.

Nine further cases published by the same writer are to be found in the *Journal of Mental Science*, vol. xlvi., pp. 719—720. The notes are very brief. It appears that at least case nine of Turner's series fulfils the desirable conditions for a diagnosis of general paralysis. Two cases were imbeciles, at least case 14 one of chronic alcoholism with delirium, and cases 8, 10, 12 and 13 probably some form of melancholia.

Wiglesworth's report contains two cases, one of "melancholia attonita," and one of "acute dementia."

(1) A woman of 48, addicted to gin, developed delusions of persecution eight months before admission, and two months later delusions of poisoning, but appeared normal to friends; one week before admission she took to bed, became weak, mute, shaky when walking; in the second week she was a little brighter but still confused, the third week she did some needlework once or twice, wandered restlessly, asked for the bill. She was feeble, had tremor of the hands, and could not protrude the tongue well. The fourth week she was restless and excited one evening. She was sad at heart, nobody cared for her, she had a child at the top of her head. She rolled on the floor and had much tremor. The sixth week she was alternately dull and talked nervously. The seventh an attack of diarrhœa came on and twitchings of tendons

and sub-febrile temperature; the eighth week the diarrhœa was arrested. The patient had a fixed expression; the arms were kept rigid, and there was occasionally a slight jerking of the tendons. Temperature normal. Death. Typical alteration of many pyramidal cells. Tuberculosis of the left suprarenal capsule.

(2) A woman of 30, married ten years, sterile and of poor health. One week after an attempt of her husband to commit suicide she became much excited, was in a panic, clutching hold of everybody, muttering words like "judgment," &c. She showed occasional tremor and in a week more or less rythmical jerks of the right arm, self-absorption, resistance, and the picture of acute dementia. Death on the nineteenth day from mere exhaustion. Wiglesworth remarks that the cell changes (inflations of nerve cells with dislocation of the nucleus) are probably analogous to those described by Charcot as occurring in the large cell of the anterior cornua of the spinal cord in so-called "infantile paralysis."

To this we add that Spiller and Hirsch have found similar changes, Spiller in the cortex of an imbecile who died with meningeal hæmorrhage (the description only shows the axonal alteration of the Betz cells; there are no Marchi specimens of the cortex, and the material was preserved several days after death), and Hirsch throughout the nervous system of an imbecile, also without Marchi impregnation.

Analysis of the Available Clinical and Anatomical Data.

On looking over this material, its heterogeneous clinical character can hardly escape attention. Taking for granted that we have good reasons, owing to the uniform findings at the autopsy, to subject all these cases to an analysis with some expectation of finding common clinical features, we meet with some disappointment at first sight. The point in which we should naturally expect most uniformity would be the physical condition, since the lesion involves mainly cells which have generally acknowledged functional attributes in the motor mechanism. In this direction as well as with regard to the mental condition and the etiological

constellation there is, however, a considerable divergence of the data.

The cases of Turner, Worcester and Wiglesworth and my own were all patients committed to hospitals for insane. Those of Ballet and Hoch died in general hospitals. Those of Ballet were cases of cachectic or alcoholic-tubercular delirium, some of them with more or less evidence of polyneuritic disorders. The case of Folsom and Hoch was one of a protracted apathetic depressive state—a more accurate picture of the general condition cannot be obtained beyond what is contained in the summary. Worcester's patient with "Landry's paralysis" began with a hypochondriacal paranoic condition, and three months later she developed the symptoms of polyneuritis, which terminated fatally in four weeks. Turner's, case 4 was one of polyneuritis accompanied by a nervous apprehensive state of one year's duration.

Turner's case 6 was one of "acute delirium" of very rapid course, and not unlike certain cases of peracute general paralysis.

My case 4 was one of alcoholico-senile deterioration of a very uneven course, also my case 8.

The great number of the cases 1, 2, 3, 5, 6 and 7 of mine, and 1, 3, 5, 7, and perhaps also 2 of Turner's, were at the outset depressive psychoses of the period of involution. In Turner's cases 3, 5 and 7, and in my cases 1 and 3, the diagnosis of agitated melancholia was made; Turner's case 1 and probably also 2 are atypical depressions, my case 2 a depressive delirium, case 5 an unusual depressive hallucinatory paranoic condition with a final deterioration resembling general paralysis, and case 6 a case of recurrent hypochondriacal depression with many features of agitation in the second attack.

After having seen the first two cases (1897) I was inclined to think that the condition present was one liable to occur on the ground of senile or pre-senile melancholia (see my foot note in the first communication), and in my differential diagnosis I depended greatly on the existence of this condition, especially when it presented somatic

deliria and rapid physical deterioration under neurological and general symptoms not unlike those seen in certain cases of general paralysis with a rapid decay, but without the typical speech defect and less definite neurological alterations. On this ground the diagnosis was made before death in the cases 4 and 5, while in case 6 even the possibility of a lesion was but vaguely entertained, since it was a second attack of a psychosis rather unlike the others. In several cases in which the general picture of the psychosis and the general condition was very suggestive shortly before death, although the motor disorders were quite vague, a diagnosis of probability was ventured upon, but with negative result, so that to-day I should put the chief emphasis on the physical findings and consider the psychosis merely as the expression of a favourable ground. It would lead too far to present here the material of negative cases, although it would prove decidedly instructive. I also omit the cases in which the histological alteration was present only in isolated cells, since these cases require more careful investigation than we have been able to give so far.

Our present knowledge can be summed up as follows. Under conditions which are not as yet clearly understood, usually in the course of certain forms of melancholia or in asthenic conditions, such as alcoholic-senile or alcoholic-phthisical deliria (and perhaps also in the course of true general paralysis?), a peculiar symptom-complex has been found to occur. With a delirious or stuporous mental condition a progressive weakness developes, usually shown first by occasional falls and a stumbling gait. This, of course, fails to be noticed in patients who are already bedridden owing to general reduction of strength or owing to stupor, or asthenic deliria, and it is in no way characteristic since it is also found in certain alcoholic deliria and in states of ordinary weakness. In most cases it is probably not correct to say that there is a paresis in the sense of well localised paralysis. The patients often present a very striking power of resistance, especially in the arms, when they do not display much strength otherwise, on account of the absence of attempts at well-

directed spontaneous motility. This condition of weakness, as Turner has pointed out admirably, is frequently, but not always, accompanied by a peculiar motor restlessness, which may vary from a blind general excitement (see Turner's case 6) to a more occasional twitching of the arms, or a subsultus tendinum. It really resembles the physical symptoms of a protracted delirium tremens, rather than the convulsive episodes of general paralysis. These symptoms are frequently very vague and, I suspect, not infrequently overlooked by attendants and physicians or merely described as weakness, or "nervous" or "hysterical" manifestations, such as tremor, peculiar motions, &c. In my sixth case, the attendant reported that the patient had occasional peculiar spasmodic twitchings in his arms, but only when he thought himself watched. The rigidity, too, which occurred at times is hardly characteristic enough. It is very difficult to distinguish it from negativistic resistence of ordinary deliria, and the pain evidenced in Hoch's case on attempts to overcome it is by no means sufficiently characteristic for any special rigidity. The description of the final episode in our case 6, and that of the more lasting condition in our case 7 and in another form in Dr. Hoch's case; and, on the other hand, the total absence of these symptoms of irritation in our case 8, show some of the extremes observed. In a few cases only (Turner, Ballet, and Worcester) polyneuritic symptoms were present. In case 7 there was difficulty in swallowing, and a partial ptosis during the last two days. There were, however, in all cases certain derangements of the *reflex-mechanisms*, either in exaggeration of the tendon-reflexes (most commonly) or absence of the knee-jerk (as in my case 5, without plain polyneuritic symptoms, and Turner's case 4 with polyneuritis). The cutaneous reflexes vary. The plantar reflex is by no means always absent. It is of interest to note that an unequivocal Babinski reflex has not been observed so far. Tests of sensibility are very fallacious, owing to the mental condition. Turner mentions the occurrence of diarrhœa during the later period of the disorder, also observed in several of our patients; our case 7 also showed irregular fluctuations of the tem-

perature without leucocytosis and without Widal-reaction; the others were not continually enough observed, so that we have only a final fever curve associated with the very common terminal broncho-pneumonia.

The vagueness of the symptoms makes it difficult to say anything concerning the *duration*. It seems to be an acute or sub-acute disorder of from two to four or more weeks, usually implanted on a more chronic deterioration. Cases like my third and fifth would almost suggest the possibility of partial remissions.

The anatomical findings. Macroscopically nothing characteristic could be noted in the nervous system. The findings are the same as those met with in cases of the same age and general condition, without the characteristic microscopic alterations. The brain weights, too, vary greatly. The membranes show various degrees of a very moderate thickening of the pia, but no adhesions, and the vessels at times varying degrees of beginning arterio-sclerosis. The first case had a scar of softening in the right caudate nucleus; no other macroscopic lesion of the brain substance was noted. In the same case there was also marked thickening of the skull with hyperostosis of the inside of the frontal bone.

Microscopical findings. The nervous system as a tissue is not markedly altered. Even the thickening of the vessels described by Turner was not present in all cases, and only few small hæmorrhages were found (as in case 1, in the cornu ammonis).

A small-celled infiltration of the blood-vessel sheaths, such as is so extensively present in those cases of general paralysis which show an anatomical lesion, was not seen in any of the cases. The neuroglia was not more abundant than in other brains of the same ages, and certainly not as it is found in general paralysis. We deal with a disorder which does not involve the tissue as a whole, but is mainly a *parenchymatous alteration of certain nerve elements* with remarkably little general reaction of the tissue. The alteration became first obvious on account of the appearance of the cell bodies, and the examination with the

Marchi-reaction showed that many fibres were in a condition of decay of the myelin sheath.

The first description quoted in the introduction mentions my first suspicion that the whole picture of the change in the cell bodies pointed to a lesion of the projection fibres, since it was typical for the "axonal alteration." It became next evident that a lesion of the brain stem was found only in the right caudate nucleus of the first case, that it was accidental and did not account for the bilateral disorder in the Betz-cells. The later cases showed no such coarse lesions, and it became further evident that the typical axonal alteration was found also in cells outside of the cortex. Similar cell changes of the character of axonal reaction had, in the meantime, been published repeatedly concerning the segmental motor neurones in nerve lesions and cases of polyneuritis and in Clarke's columns of cases of tabes and meningitis, and I had drawn attention to the same change in these and several other cell types under less characteristic conditions in my "Critical Review," &c. The examination with the Marchi method yielded positive results in the hands of Turner and Hoch. But when it was applied to our case 6, it showed a marked discrepancy between the number of fibres which have the reaction of degeneration in the pyramidal tracts, and the number of cells with axonal alteration, and this led to a more extensive examination of my cases 7 and 8.

The result shows conclusively that it would be wrong to see in it only a disorder of the cerebral cortex alone. It is a diffuse affection, mainly of elements within the neural tube, but undoubtedly also present in some of the neurones constituting the "peripheral nerves." In several cases (Turner, Worcester, Ballet) the polyneuritic features were indeed quite prominent.

The general histological character of the lesion is the same as what is described in parenchymatous peripheral neuritis or degeneration. From the standpoint of general pathology, we see in this a degenerative process, analogous, but of course not necessarily homologous, with degenerative processess of other cell types, but considerably modified, owing to the

peculiar structure of the tissue elements of the nervous system, and especially of the elements most directly under consideration, the nerve-elements or neurones, with their highly differentiated cell bodies and processes and medullary sheaths, &c.

There is, unfortunately, no general term available for this condition. The most common distinctions are those according to localisation. From the "preneuronic" period, we have a very crude way of dividing the nervous system into brain, spinal cord and peripheral nerves. In the peripheral nervous system, we speak of peripheral neuritis, a vague term which includes the reaction to traumatic and infectious and toxic results. Like the type under discussion above, the *parenchymatous peripheral neuritis* does not involve so much the coarse anatomical structure "nerve," as the individual nerve fibres; and when we speak of parenchymatous neuritis of certain sets of nerves, we imply by it a degenerative affection of more or less of the nerve fibres which help constitute the "nerve."

In the central nervous system this condition has attracted attention, mainly in connection with a few chronic diseases of the spinal cord. There it is spoken of as systemic and non-systemic degenerations, involving a more or less great number, either of fibres of a recognised set, or of several sets, or diffusely scattered without any regard for a limitation to such sets. The systemic and polysystemic degenerations in the cord have received their special terminology, usually derived from the clinical picture, and as long as only few "systems" were usually considered, the tracts with degenerations were easily enumerated. For the diffuse degenerations found, for instance, in pernicious anæmia, a more definite description was not attempted, beyond a general localisation of areas in the cord section.

The difficulty makes itself much more felt in those who work on the brain, in which system diseases have been relatively little described outside of the typical secondary degenerations; and those who work with some forms of the neuron concept have additional reason to be dissatisfied with the existing terminology because we cannot stop

at the study of bundles of fibres; we must take into consideration that the fibres are parts of cells, and that the correlation of cells and fibres is a prominent postulate of neurological accuracy. The conditions which occurred in our cases illustrate this well. At first we discovered a change in the cell bodies, and now we know, moreover, that there are special sets of corresponding fibres in degeneration. In polyneuritis the degeneration of the myelin sheaths was the first finding; and the cell changes became known later. As we shall presently see the change in the cell is quite characteristic; but its nature, and the nature of its correlation with the alterations of the fibre is hardly explained, not even with the most high-sounding theory.

In a discussion on Landry's paralysis, the writer has already pointed out in 1896, how vague the terms "polyneuritis, myelitis, and encephalitis" are when we deal not with the local disease of the entire tissue of a locality and its remote consequences, but with more general toxic, infectious or asthenic parenchymatous degenerations in the nervous system. The present experience shows that the traditional nomenclature based on coarse anatomical localisation is inadequate for description and didactic purposes. Many cases of peripheral neuritis of a not local origin are probably peripheral only because the other parts of the nervous system are not examined with equally adequate methods as the peripheral nerves. To judge from a rather large material treated with the Marchi method, the extent of decay of medullary sheaths throughout the nervous system is far greater than the old hæmatoxylin methods made one suspect. Unfortunately it is difficult to find a term which would apply indifferently to all degenerated conditions with decay of myelin sheaths of any nerve unit. I propose to adhere to the shortest word, "neuritis." It should not meet many objections, because the concept "inflammation" is more and more dissolved, and one would hardly see anything "inflammatory" behind the syllable "itis." But the terms might seem to refer especially to nerves, and their next analogues, nerve fibres. The very

persistence with which the attribute "peripheral" reappears with the word neuritis shows, however, that the use of the term is not as fixed that we should not be allowed to speak of "central neuritis," or in more general conditions of "general neuritis," instead of a long descriptive expression, "diffuse degenerative affection of nerve elements with myelin decay and axonal reaction." It seems probable that the rising generation, with the increased knowledge of the life or nerve elements or neurones acquired since Forel's inspiring paper of 1887, will think more in terms of nerve elements than the generation from which we received our training, and also some of our prejudices. They will get accustomed to think more of the histologically important correlations of parts than of coarse anatomy whenever they are not especially anxious to refer to topographical conditions, such as encephalitis, myelitis, and neuritis of local origin.

There is another feature which we usually think of in connection with the terms neuritis or polyneuritis. We can safely say that no diffuse parenchymatous neuritis is truly general. There are—clinical experience shows this—fairly definite types of distribution more or less characteristic for the origin, so that even in the great diffusions there is usually a certain system. On the other hand, the more accurate methods have shown that system diseases are rarely strictly monosystemic. Tabes does not limit itself absolutely to the intraspinal course of the afferent segmental neurones; amyotrophic lateral sclerosis is not merely a degeneration of the pyramidal fibres, &c. The cases under consideration are classed under the name of *central neuritis* with the understanding that we deal with a parenchymatous degeneration of nerve elements, *mainly* of the *central* nervous system, and that this type is one belonging to the general group of "diffuse neuritis" with a certain degree of systematisation.

Concerning the extent of the central neuritis, I refer to the cases 7 and 8, which show that there are various degrees of the disorder and perhaps also various distributions.

We cannot omit the discussion of a rather difficult point

in the problem, namely, the peculiar distribution of the changes within the same nerve elements. This, unfortunately, is a question in which we ought to have at our command a larger material and working force than our State hospital furnishes to-day. Many of the conflicting statements one meets in a digest of the pertinent literature come from the looseness partly of the nomenclature of the writers, partly of the descriptive evidence furnished, so that perhaps the problem is not quite as hopeless as it appears. In my "Critical Review" I pointed out how small our chances are under the best opportunities to really get a sufficiently definite idea of the condition of any entire nerve element, and this makes it especially desirable that results should not pass into the literature without a careful analysis of the validity of the facts as well as the descriptive terms and the definitions adhered to.

As an instance, I quote the very article in which Faure communicates his six cases of cell changes in the cortex in toxic infectious mental disorders quoted above (p. 95). He discusses the question whether the cell alteration is always secondary. Several of his cases had plain polyneuritis, but he used only alcohol methods in the cortex and not Marchi's method, and he remains wisely reserved. He merely adduces one argument against the necessarily secondary character of the cell alterations, which might well be criticised in a few words. Together with Ballet, he sectioned the projection fibres underneath the motor cortex in seven dogs and killed them eight, eleven, eighteen and twenty-one days after the operation. "Comparison of the sections of cortex from the operated and normal side demonstrated plainly the disappearance of the large pyramidal cells." In three cases they also examined with Marchi's method without finding any alteration in the fibres—hence there is a discrepancy with "the complete disappearance of the large pyramids"—or have the fibres too completely disappeared in three weeks? Only one case of the seven (and that one was complicated by infection) did present the axonal alteration (instead of the "disappearance"?). These contradictions invalidate the weight of the communication

very seriously and relieve our feeling of standing before another great puzzle. His statements are so flatly at variance with what Bregmann and others have shown and also our own experience concerning the cortex in subcortical lesions, that we are forced to suspect a serious error of observation, and that we regret to see the article quoted without criticism of this glaring contradiction.

A further complication lies in the indiscriminate use of the term "chromatolysis." The expression came into vogue when the stainable portion of the cell body and of the dendrites were newcomers in neurological descriptions, and, perhaps, absorbed undue attention. Since then it has again become evident that in the physiological and pathological life of nerve-cells it is wrong to classify all the changes according to the condition of only one part of the cell. While we cannot claim to be anywhere near a final recognition of the possible changes and their classifications, we are able to-day to speak at least of a few *types* with a possibility of being understood by those who take the trouble to acquaint themselves with well-described distinctions. The most striking types are those of pyrexia (commonly called acute alteration), the axonal reaction present in our cases, Hoch's shrinkage-alteration, the mere rarification of the stainable substance, &c. To classify these as varieties of chromatolysis promotes the existing confusion. The term "central chromatolysis" is utterly misleading, since there are at least two such types, the pigmentary and the axonal reaction, and because it suggests a change *around* the nucleus. By *axonal reaction*, we understand that cell alteration which we can produce experimentally by the section of the axone. It is as it were a focal disorder within the pericaryon; splitting up and dissolution of the stainable bodies usually between the place of origin of the axone and the nucleus (not *around* the nucleus), swelling of this region with involvement of the greater part of the cell-interior and dislocation of the nucleus towards the surface and even beyond the normal contour; persistence of a peripheral ring of lumps, and of the spindles in the dendrites. Decay, or at least fatty alteration of the medullary sheath of the axone is

a frequent accompaniment. When this alteration is combined with acute alteration (see p. 108) the stainable bodies of the periphery and dendrites are also dissolved, and the dendrites showed the characteristics of the acute alteration. When we speak of "axonal alteration," we imply that this is the *type* identical with that always observed where section of the axone occurred near enough the cell body to produce any reaction at all; and this *type* we have now found to exist where the nerve fibre was merely diseased as it was already found in polyneuritis, and also under conditions in which disease of all the nerve fibres in question has not been demonstrated, but where at least some fibres show the presence of the general conditions for degeneration of fibres (my case 8 and the instances mentioned in p. 94). It would seem from our cases that the cell body may show the alteration before the fibre is visibly affected, unless we should assume the other possible alternative that many of the *débris* of fibres were already resorbed; and Warrington, Goldschieder and Marinesco claim that the same change may occur if a set of superimposed neurones is affected which is normally the source of stimulation of these cells. It is obvious that the question is still complicated; but this should be a special reason to make the distinction of the type which is already recognised, instead of throwing it together into the hazy "chromatolysis." The axonal reaction to us is an, as yet, unexplained complex of manifestations, which as far as we know now, seems closely related to the constellations under which we recognise also myelin decay. Not all the cell types react equally, and the assumption of a "central neuritis" is probably justified only where entire sets of cells show the reaction without there being any disease of the *tissue* which would account for the occurrence of degeneration after the type of secondary and retrograde degeneration. The frequent occurrence in Clarke's columns and in other cell types requires further studies, and we must admit that we are still far from knowing all the possible histological steps in the process.

The *nosological position* of this condition is as little within the reach of definite decision as the mental disorders

which formed the beginning, or as little as other not directly toxic forms of polyneuritis; and this is not the place to rehearse the reasons why we prefer a statement of ignorance to a discourse on auto-intoxications at this stage of "mental pathology." We propose to wait or work for more tangible data, and commit ourselves to the following remarks.

In order to obtain the necessary amount of data concerning frequency of the central parenchymatous nerve decay, we need a larger number of thorough examinations also of the *supraspinal* nervous system in many types and conditions of nervous diseases. I refer here to such possibilities as studies of cortex in polyneuritis or more or less diffuse spinal affections, such as were described by Russell, Batten, and Collier, under the name of subacute combined degeneration of the spinal cord, and to such lines of work as the studies of Zappert on the root and cell changes in the central nervous system of the child, the studies of Kirchgässer on the nervous system in cancer, cachexia, &c., and the publications of Luce. My own experience in the study of the nervous tissues, with modern methods for the observation of the cells as well as the fibres, cautions me against hasty statements. I merely venture to make the remark that our ideas of the normal nervous system, on the one hand, and of what might be called fatal true degenerations on the other, represent extremes, and that our present delicate methods show us stages to which the standards of text-book correlations obtained from less discriminating methods—viz., the law that degeneration implies abolition of function and, probably, irreparability, if found in the central nervous system—should not be applied without much care. There are most probably various degenerative conditions in the central nervous system, not all of clearly-known clinical significance, and some of them may be functionally trivial, and it is quite probable that the Marchi method does not merely show old facts in a more precise garb, but certainly also conditions of less striking importance than the serious degenerations of the more primitive neuro-pathology, which saw degeneration only when it was

macroscopically visible. It is especially deplorable that studies of the axones are so difficult in the central nervous system, since the recognition of a disorder of the axone should be furnished in addition to the one of its myelin sheath, before the relation between function and lesion should be discussed. My studies in this direction are hardly mature for publication.

Findings similar to ours have lately been established by Bonhoeffer and by Starlinger. Starlinger describes, in his "Beitrag zur pathologischen Anatomie der progressiven Paralyse" (Monatsschrift of Wernicke and Ziehen, vol. vii., p. 1-13), the presence of fairly-localised degenerations which do not differ much in their general distribution from what we found in our case 7, were it not for their more unilateral occurrence and the difference in the clinical history of the cases and in the condition of the cortex, which Dr. Starlinger kindly demonstrated to me. The latter shows the correctness of his diagnosis with just as much certainty as we should exclude the typical lesion of general paralysis in our own cases. The distinction is probably in the main as follows:—The predominant character of general paralysis is that of a tissue alteration, whereas our findings are strikingly devoid of this feature. According to Starlinger, the distribution of the degenerations in general paralysis is dependent on the extent and localisation of tissue alterations, and it is, therefore, less systemic and less symmetrical. Besides this process, there *may* exist in general paralysis one allied to the type of central neuritis, as case 9 of John Turner's series might suggest. Personally, I have not met a case of true "central neuritis" in clinically and anatomically plainly established general paralysis.

The findings of Bonhoeffer refer to alcoholic delirium (Wernicke's Psychiatrie) and to "Seitenstrangerscheinungen bei acuten Psychosen" (Psychiatrische Abhandlungen, Heft 2, Breslau, 1896). The latter, although observed clinically only, suggest strongly a more frequent occurrence of central alterations than has been commonly surmised, and form a reason why we prefer to be rather conservative as to conclusions. The alcoholic degenerations found by Bon-

hoeffer are most likely akin to those in our own cases, and we can confirm the observation from a case of plain alcoholic delirium in which, however, the lesion was in no way as extensive as in those of "central neuritis" of non-alcoholic origin described above and in which the cell alteration was far from general.

Turner, in his first description, calls our cases a "form of dementia associated with a definite change in appearance of the pyramidal and giant cells of the brain." Since the above was written he has even ventured to base on these cases his theory "concerning the physical conditions of the nervous system which are necessary for the production of states of melancholia, mania, &c." While we have seen that most of our cases have arisen on a basis characterised by depressive states, we have met at least two cases of alcoholic-senile deterioration in which depression was absent or trifling; and in a sufficient number of cases we have failed to establish all the findings in undoubted instances of profound depression. Neither the expression "dementia" nor the inferences as to melancholia and mania appear to me warranted at this stage of our observation. Hypotheses have a value only if they stimulate to further research, but not if they give premature satisfaction. For this reason I prefer to call the general condition of our patients by the terms "delirium," "stupor," and "coma"—not by the term "dementia," simply because I consider it impossible to establish what deserves the name "dementia" in any of these states, as they prove fatal before an examination for dementia is quite conclusive. We might as well call a more or less profound delirious and stuporous condition of the fever-alterations and of certain intoxications a "dementia." Another question is the relation of these mental states to the preceding melancholia or alcoholic or senile delirium. Here, too, reserve is advisable. As long as we do not know the conditions from the point of view of entities fulfilling the requirement for an admission into true causal chains, we do best to treat them merely as empirical chains of facts.

That the modern views even on "general paralysis of the insane" to-day appear to be in a harbour of dogmatic

security, is chiefly due to their plausibility and their value in practice and teaching; yet even they would hardly be able to stand a moderately severe criticism unless we modestly admit their empirical character. The discussion has merely been stopped owing to the difficulty of getting at more accurate facts. For this reason we avoid a long discussion of the question whether our group of cases should be called a type of general paralysis.

Summary.—(1) Eight times in 200 autopsies in which a microscopic inspection of the cortex took place a condition of bilateral changes of the nature of the axonal reaction in practically all the Betz-cells was observed, accompanied by the same changes in other cell types and, where this was looked for, by decay of the medullary sheaths of some of the corresponding sets of fibres, whereas the characteristic disease of the tissue of general paralysis (gliosis, vascular infiltration, &c.) was absent. An extensive examination of large areas of the brain in three cases shows the existence of a partially systemic but widely distributed and strikingly symmetrical parenchymatous alteration of numerous nerve elements, chiefly of the cortico-thalamic connections of the motor area, the auditory radiation, the forceps, the pyramids, the fillet, the restiform body and, to a lesser degree, the posterior columns of the cord, the intersegmental elements, and the segmental efferent motor elements. The change is that of axonal reaction in many of the large cell types and decay of the myelin sheaths. Instead of the long descriptive term, "Partially systemic parenchymatous degeneration principally of the central nervous system," we propose the expression, "central neuritis" in the sense of an equivalent of parenchymatous neuritis, but mainly of central distribution.

(2) This alteration has been found to occur in peculiar forms or end-stages of depressive disorders, near or after the climacteric period, alcoholico-senile and alcoholico-phthisical cachectic states, idiocy, and perhaps also general paralysis (Turner's case). Ordinary infectious and cachectic states do not, however, appear to form an important link in the causes.

(3) Clinically, the symptoms are rather vague: after a

course in which there is no suspicion of organic disorder, there appears, more or less suddenly, difficulty of locomotion, increasing weakness for coordinated movements, at times jactations of the limbs and rigidity and disorders of the reflexes, often together with diarrhœa and occasional febrile fluctuations; the mental condition in this terminal episode is either that of anxious perplexed agitation, delirium, or stupor, similar to a protracted delirium tremens. The nature of the disorder appears to be an equivalent of the general type of parenchymatous nerve-cell alterations, but, in distinction from the well-known infectious and toxic types of "polyneuritis," of pre-eminently central distribution.

BIBLIOGRAPHY.

(1) WIGLESWORTH, JOSEPH. "On the Pathology of Certain Cases of Melancholia Attonita, or Acute Dementia," *Journal of Mental Science*, October, 1883, p. 355.
(2) MEYER, ADOLF. "Demonstrations of Various Types of Changes in the Giant-cells of the Paracentral Lobules," *American Journal of Insanity*, vol. liv., pp. 221-226, October, 1897.
(3) WORCESTER, W. L. "A Case of Landry's Paralysis," *Journal of Nervous and Mental Diseases*, 1897.
(4) FAURE. *Revue Neurologique*, December, 1899.
(5) TURNER, JOHN. "Note on a Form of Dementia, &c.," BRAIN, 1899, vol. xxii., pp. 575-585.
(6) *Id.*, *Journal of Mental Science*, vol. xlvi., pp. 719-720.
(7) SPILLER. *Journal of Nervous and Mental Diseases*, 1899.
(8) HIRSCH. *Journal of Nervous and Mental Diseases*, 1898.
(9) MEYER, ADOLF. "Critical Review of the Data of Neurology," *Journal of Comparative Neurology*, 1899.

EXPLANATION OF THE FIGURES.

FIG. 1.—Betz-cell from *Case* 1. Axonal reaction. $\frac{1}{12}$ oil immersion, Leitz.

FIG. 1*a*.—Betz-cell from the same case. Vortex conditions of the chromophilic portions. $\frac{1}{12}$ oil immersion.

FIG. 2.—Betz-cell with vortex formation, *Case* 8.

FIG. 2*a*.—Large pyramid of the frontal cortex (*Case* 8), with axonal alterations. $\frac{1}{12}$ oil immersion.

FIGS. 3 and 4 (in text).—Right hemisphere of *Case* 7, with the levels of the slices for Marchi specimens: (*a*) through the foot of the frontal lobe;

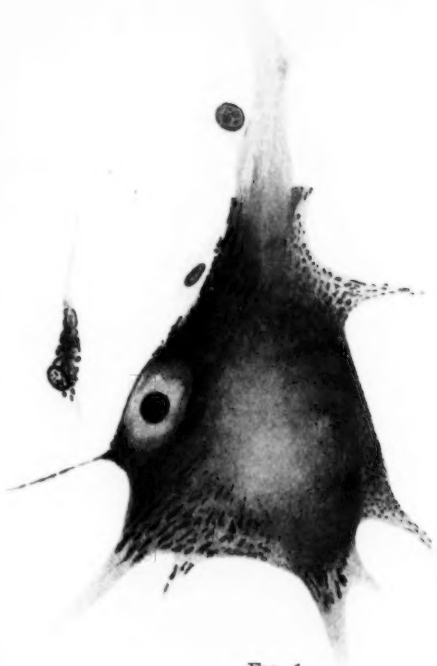

FIG. 1.

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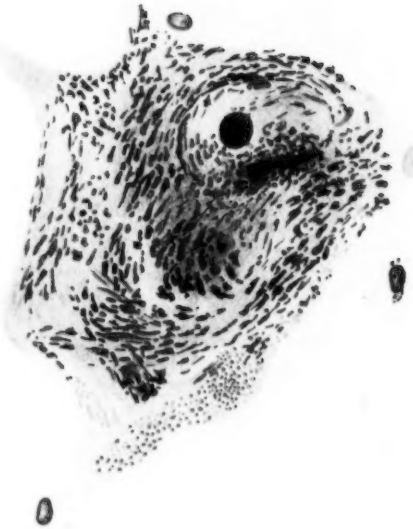

FIG. 1*a*.

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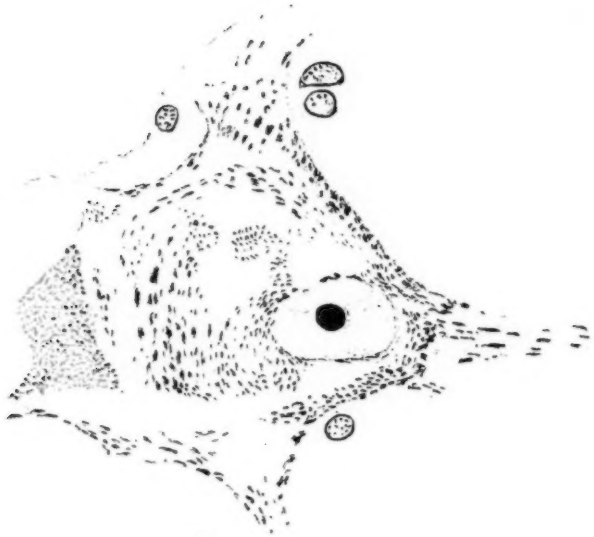
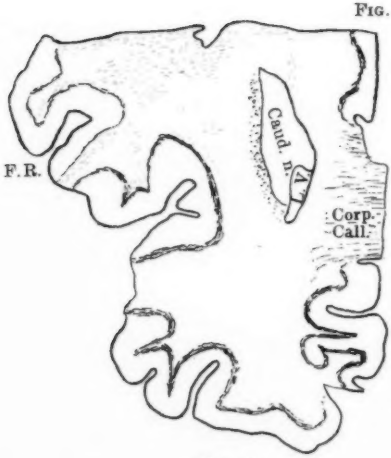


FIG. 2.

FIG. 2*a*.



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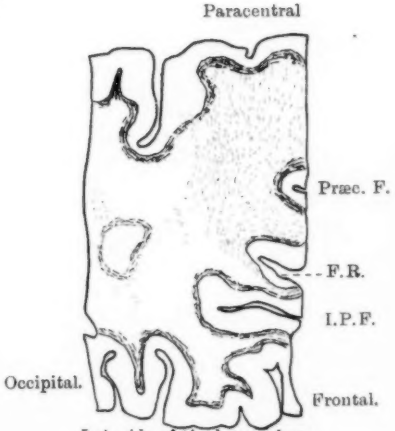

Lat. side of the hemisphere.

FIG. 6.

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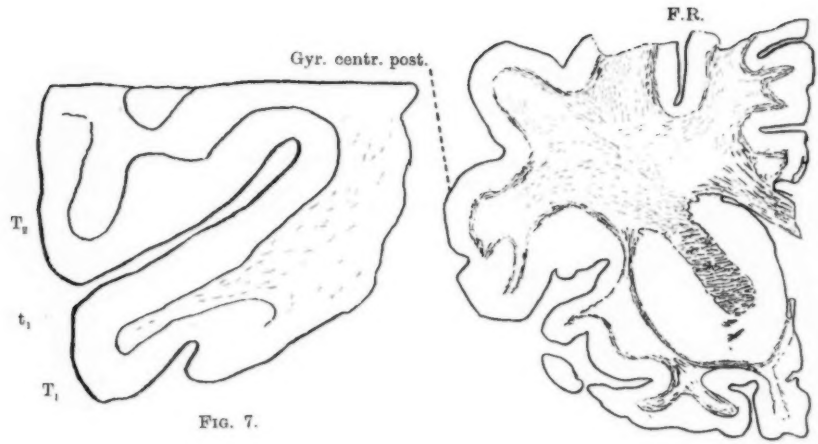
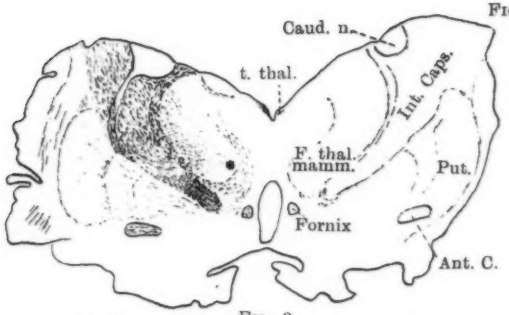


FIG. 7.

FIG. 8.



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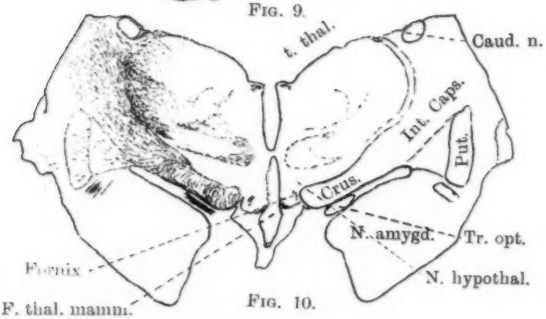

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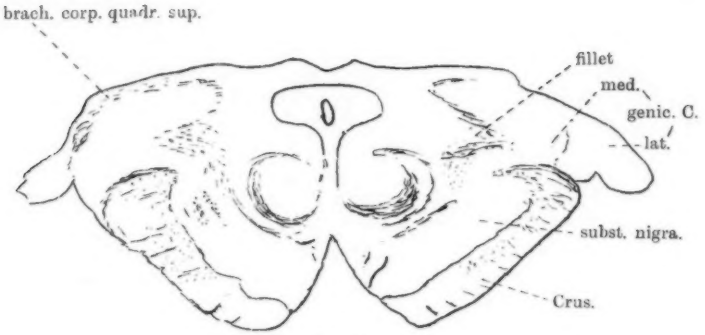

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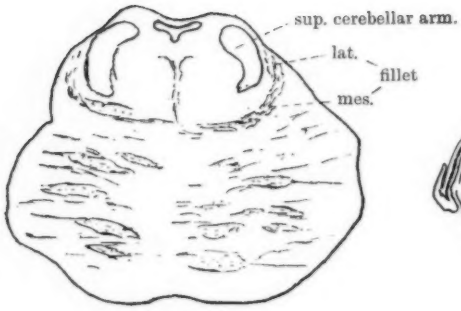

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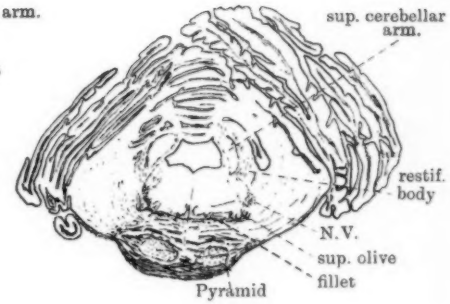

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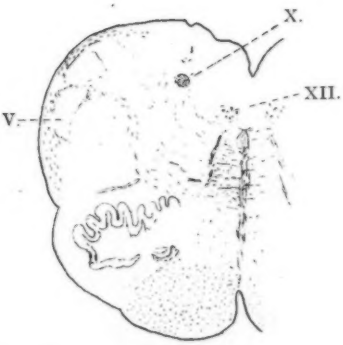

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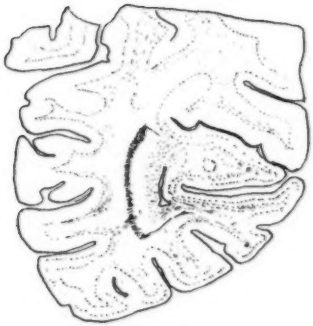

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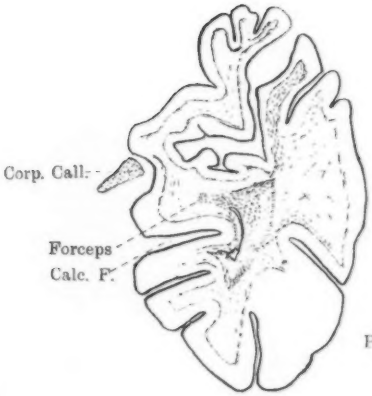

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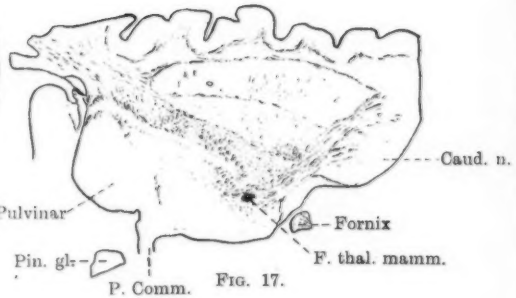

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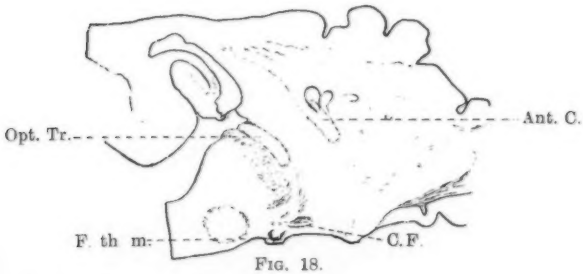


FIG. 18.

Adolf Meyer del. *Bale & Danielsson, Ltd., Lith.*

(*b*) underneath the paracentral lobe; (*c*) through the temporal lobe; (*d*) frontal section through the motor area and in front of the anterior commissure.

FIG. 5.—Section from fig. 3*a*. Degenerating fibres limited to the motor area and its part of the centrum ovale, and of the corpus callosum.

FIG. 6.—Section from fig. 3*b*. Degenerating fibres limited to the projection system of the motor area. Only few fibres from the parietal lobe.

FIG. 7.—Section from fig. 3*c*. Degenerating fibres limited to T_1.

FIG. 8.—Section from fig. 3*d*. Degenerating fibres from the motor cortex to the dorsal part of the internal capsule and the corpus callosum.

FIG. 9.—Section from the anterior part of the thalamus (only the left side drawn; the whole being symmetrical). Degeneration of fibres of the internal capsule and its radiation into the lateral thalamic nucleus only.

FIG. 10.—Section from a posterior level. Division of the degenerating fibres into a radiation of the ventro-lateral nucleus of the thalamus and pyramidal fibres.

FIG. 11.—Mid-brain, with degeneration of pyramidal fibres only in the crus, few fibres in the fillet, and few fibres in the arm of the superior corpus quadrigeminum.

FIG. 12.—Section through the posterior corpus quadrigeminum. Degenerating fibres in the pyramid and fillet.

FIG. 13.—Section through the trigeminal segment. Degenerating fibres in the pyramid, the corpus restiforme, and few in the fillet.

FIG. 14.—Section through the pneumogastric segment. Degenerating fibres in the pyramid, the corpus restiforme, and few in the fillet.

FIG. 15.—Section from the left occipital lobe. Degeneration practically limited to the forceps and its radiation.

FIG. 16.—The same on the right side, with evidence of degeneration in the splenium of the corpus callosum.

FIG. 17.—Horizontal section through the left thalamus of *Case* 8. Degeneration limited to the internal capsule and the radiation of the anterior half of the lateral thalamic nucleus; scanty degeneration in the radiation of Gratiolet.

FIG. 18.—Section parallel with the last one. Very scanty degeneration, limited to the pyramidal tract.

SECTION OF THE POSTERIOR SPINAL ROOTS FOR THE RELIEF OF PAIN IN A CASE OF NEURITIS OF THE BRACHIAL PLEXUS; CESSATION OF PAIN IN THE AFFECTED AREA; LATER DEVELOPMENT OF BROWN-SEQUARD'S PARALYSIS AS A RESULT OF LAMINECTOMY; UNUSUAL DISTRIBUTION OF ROOT ANÆSTHESIA; LATER PARTIAL RETURN OF SENSIBILITY.[1]

BY MORTON PRINCE, M.D.

Physician for Nervous Diseases to the Boston City Hospital (U.S.A.).

THE report of this case is published because it shows:—

(*a*) The areas of anæsthesia following section of the fifth, sixth, and seventh cervical spinal roots.

(*b*) The relief of pain as a result of the operation.

(*c*) Development of Brown-Sequard's paralysis following laminectomy.

(*d*) Partial return of sensation in previously absolutely anæsthetic areas.

Jeremiah F., as a result of being struck by a railroad train, received a Colles' fracture of the left wrist, and developed a complete and absolute paralysis of nearly the whole left arm. The paralysis was typical in its distribution, in its course, in the electrical reactions (R.D.), atrophy, &c., of a neuritis of the brachial plexus. The paralysis has continued ever since, and the arm has been useless from the shoulder down. The absolute paralysis involved all the muscles of the shoulder, upper arm and forearm, but the intrinsic muscles of the hand and the pronators of the forearm were only partially paralysed. There was diminution of sensation over limited areas, which will later be described. The most intensely affected muscles were the upper arm group, namely, the supra and infra spinati, deltoid triceps and bicipital group, for the absolute paralysis of these muscles continued at

[1] Read at the meeting of American Neurological Association, May, 1900.

the end of a year and a half after the injury, with marked atrophy and loss of the electrical reactions, while at the end of a little over a year some power had returned in the forearm muscles, for at the end of this time Jerry could flex and extend both the wrist and fingers, though with very slight strength. There was also slight power of pronation. From this it will be seen that all three cords of the brachial plexus were involved. The most diseased fibres were from the fifth, sixth, and seventh cervical segments, while those coming from the eighth cervical and first dorsal, supplying the intrinsic hand muscles and the long flexors and extensors of the fingers, were partially implicated. The heft of the injury was therefore to the fibres that make up the supra scapularis, musculo-cutaneous, circumflex nerves and triceps portion of the musculo-spiral nerve, and therefore to that portion of the plexus which is made up of fibres from the fifth, sixth, and seventh cervical. At any rate, the fifth, sixth, and seventh cervical fibres were most severely injured, while of these three the fifth and sixth suffered the most.

From the time the splint was taken off Jerry suffered most intensely from pain which was located chiefly in the thumb and forefinger. For this he was referred to the Clinic for Nervous Diseases, Boston City Hospital, where he was under treatment for a long time. For the relief of this pain, which was exceedingly intense and justified almost any operation that promised relief, three different operations were undertaken at different times, but none of them seem to have been based on sound neurological principles. About two months after the accident the radial was stretched in the forearm, under the theory that the pain might have been due to injury of the nerve at the wrist by the fracture; but the operation failed to give relief. About seven months later he was operated on in another hospital. The exact nature of the operation could not be ascertained, but an incision was made along the course of the nerves in the upper arm, it is said with the idea of removing a "callus." Later still, about fifteen months after the accident, he again entered the surgical ward of the City Hospital, and a second incision was made in the upper arm, in the region of the brachial plexus, and what was thought to be a piece of diseased nerve removed, but which turned out to be a piece of fibrous tissue. Shortly after this (October, 1899), he was again referred to me by Dr. J. C. Munro, in whose service he was at the time. At this time the paralysis was as has already been described, and the arm was perfectly useless for practical purposes. Whatever strength had been regained in the hand could not be utilised because of the intense

pain in the thumb and forefinger already spoken of. A rough touch to these digits caused the most excruciating and unbearable pain. The hand and arm had to be handled with the greatest care, and was carefully nursed and kept supported by the patient. He expressed himself as willing to undergo any operation for

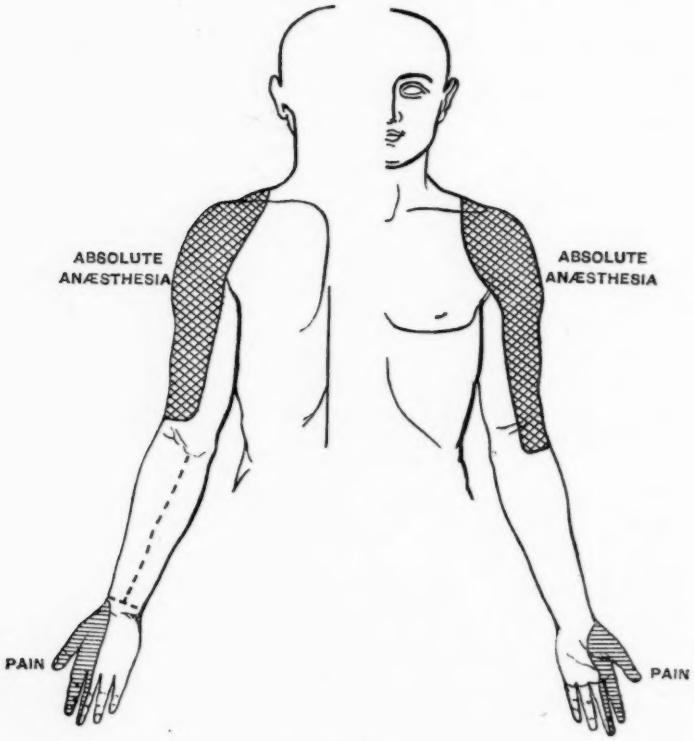


DIAGRAM I.

Showing location of pain in the hand prior to operation; also area of absolute anæsthesia at same period.

relief. The distribution of the pain may be seen by reference to diagram I. It was most intense and disabling in the thumb and forefinger and corresponding half of middle finger, back and front, but there was another line of pain of much less intensity and

importance extending like a half-bracelet around the back of the wrist, and from the middle point of this bracelet up the middle of the forearm to the olecranon. It will thus be seen that the severe pain in the thumb and forefinger did not correspond in distribution to the area of any nerve supply, but did correspond to the usually accepted distribution of the sixth cervical root or segment.

I accordingly advised as a rational procedure, and the only one likely to give relief, section of the posterior roots of the fifth, sixth, and seventh cervical nerves, the fifth and seventh being included in case some of these fibres overlapped in the affected area, and therefore might be the source of the trouble. Laminectomy was accordingly done by Dr. J. C. Munro. The operation was a very severe and bloody one—one of the most difficult I ever witnessed, in consequence of the fact that the patient was a thick-set man with a "bull neck." From the moment of coming out of the ether Jerry was completely free from pain in all the previously affected areas, the wound healed perfectly by first intention, and the operation was a success for the purposes for which it was undertaken.

Jerry's troubles, however, did not end here. After he began to get about it was noticed that he exhibited a limp and weakness of the left leg, and examination showed that there was a moderate Brown-Sequard paralysis. The left leg was decidedly spastic and paretic. There was a marked increase of the deep reflexes of this leg including an ankle clonus, and he could not raise his weight on the leg, while there was a moderate diminution of sensibility over the whole right leg. This anæsthesia was limited above by the groin; on the left sensation was normal, as were muscular power and the reflexes on the right. During the operation the danger of a hæmorrhagic clot being partially formed within the spinal canal occurred to me, as indeed it had at previous operations of this character, although I had never known it to occur. I may say here, merely to emphasise this point, that I called attention to this danger at the time of the operation. Now this possibility was suggested anew by the occurrence of the paralysis. Nevertheless, it is equally likely that the paralysis was due to pressure from granulation tissue or some form of callus. As the exact time of onset of the Brown-Sequard paralysis is not known, it is not easy to decide between the two possibilities. That the paralysis is due to pressure of some sort and the sequence of the laminectomy is scarcely questionable. It is noteworthy that the pressure on the cord is on the side (left) on which the roots were cut.

But still more trouble awaited Jerry. There gradually developed severe pain on each side of the neck behind, so that turning the head to either side caused great pain, of which he complained considerably. Thus it happened that at the time of his discharge from the hospital, about three months after the operation, he still suffered from severe pain, though in a different region from that originally affected. The cause of the pain in the neck was undoubtedly a neuritis, due to some nerve being injured either by the operation or by the healing thereof. Although the arm itself was free from pain, it was perfectly useless, and the patient expressed a strong desire to have it amputated to get it out of the way. He has been promised a consideration of this request if, at the end of another year, he has regained no practical use of it.

A recent examination of Jerry (April 10, 1900), about five months after the operation, shows a great improvement in his condition, and that the operation has been a success for the purpose for which it was undertaken. He is completely free from pain in his hand and arm, and the pain from the neuritis in the neck has also ceased. He can turn his head freely without discomfort. He is now free from pain so far as his original trouble is concerned, but he does have pains about the shoulder, which apparently are due to the weight of the paralysed arm dragging upon the parts. By supporting the arm this pain is relieved. But the arm remains completely paralysed from the shoulder down, and is absolutely useless. The paralysis of the hand has rather increased, so that he can little more than move the fingers. The electrical reactions have almost completely vanished. On the other hand the Brown-Sequard paralysis has improved very much, so that he walks without perceptible limp, although the left leg is still weaker than its fellow; he can go up and down stairs only with care, and he cannot raise his weight by his left foot or leg. There is still moderate anæsthesia in the right. The limiting line of the anæsthesia is at the height of the groin as before. An uncomfortable symptom, and one of some interest, is a sensation of intense coldness in the right foot. He still desires to have the arm amputated.

Anæsthesia.

The distribution of the anæsthesia following the section of the spinal roots was very interesting. It differed materially from that which would be expected from the diagrams of Starr, Kocher, and Wichmann. It was studied with great care.

PHOTOGRAPH A.

PHOTOGRAPH B.

In the forearm and hand the area (radial aspect) included within the heavy lines is that in which absolute anæsthesia developed after section. Above it joined the previously anæsthetic area in the shoulder. The dotted area represents diminished sensibility on the back of the forearm. This existed previous to operation. The broken line over the shoulder indicates the degree of return of sensibility, April 10.

Several charts of the anæsthetic areas were taken at different periods previous to the final operation. They all were in substantial agreement excepting that the chart taken just before the section of the posterior roots showed a new area of absolute anæsthesia (diagram I.) covering the

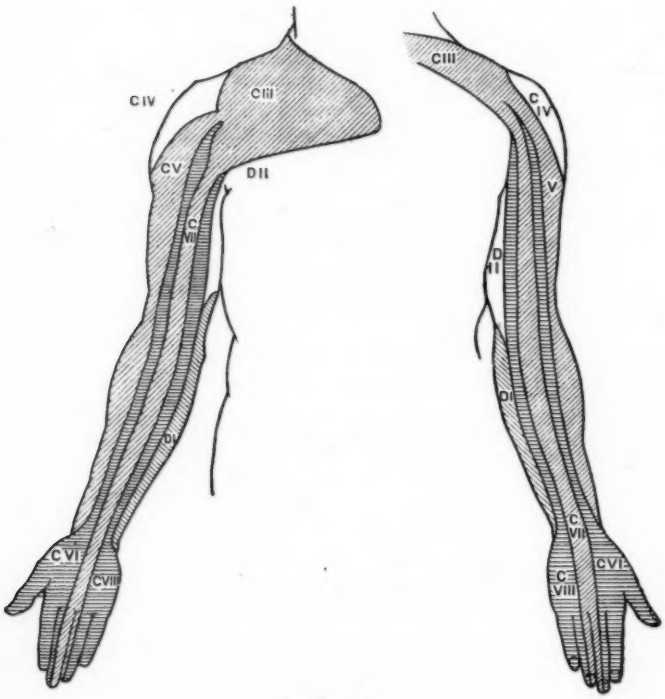


DIAGRAM II.

After Starr. To show cutaneous root-areas over front and back of arm and hand.

shoulder and extending above the clavicle about half-way up the neck, and below over the corresponding upper two-thirds of the arm. Whether this was due to one of the operations, or to an extension of the neuritis, is not clear. Its presence was important only so far as it made it impossible to deter-

mine the upper limits of the distribution of the fifth root, as the anæsthesia from the latter merged into this already existing anæsthetic area. Besides this, there was in the forearm and lower third of the upper arm, previous to section of the roots, and as a result of the neuritis, an area of impaired sensibility. This impairment was limited to the radial aspect, and was continued down over the thumb and forefinger. It included both pain and touch, but not in coextensive limits. As analgesia it extended over the whole of the thumb, but only over the back of the forefinger and back of the hand. As tactile anæsthesia, it spread over the thumb and palmar aspect of the fore and middle fingers, and a portion of the back of the hand.[1]

But, with the exception of that over the shoulder, neither the analgesia or the anæsthesia was anywhere absolute. For the most part, the anæsthesia consisted of numbness, that is to say, the patient *felt a light touch*, but the feeling was described as "numb." The analgesia was deeper than the tactile anæsthesia, but as tested by a pin prick was not absolute. It was most intense in the *whole* thumb, back of hand and back of the forefinger, that is, those areas of the hand which (excepting the forefinger) became absolutely anæsthetic after section of the roots. The difference between this slight or moderate impairment of sensibility and the absolute loss of appreciation of touch and pain which in parts of the same areas followed the section of the spinal roots, allows us to recognise the cutaneous distribution of the latter.

According to the usual description (diagram II.) of the root areas, the absolute and complete anæsthesia, after section of the roots, besides including the radial side of the arm (V.), should have covered the thumb, forefinger and adjoining half of the mid-finger (VI.), with only impaired sensibility, perhaps, over the remainder of the mid-finger and adjoining half of ring finger;[2] this latter because the area of

[1] It is interesting to note that this total area corresponds fairly closely with the areas of the fifth, sixth, and seventh roots as given by Starr, Kocher, and Wichmann. The diagrams of these writers, however, differ considerably in the limits of the areas allotted to each of these three roots separately.

[2] Kocher includes even the ring finger in the sixth root area.

the seventh would have received additional fibres from the adjoining eighth cervical or first dorsal. This is not, however, exactly what happened. *The absolute loss of sensation for touch and pain was limited to the whole thumb with its ball, back of the hand and a strip along the radial aspect of the forearm and arm* (see photographs). Over the other fingers the impairment was not perceptibly increased over the slight numbness that was present before operation; excepting that over the backs of the proximal phalanges there was a zone (dotted in photograph) of diminished sensibility for pain and touch, which was not noticed before the operation, and over the whole back of the forefinger the analgesia was increased, but tactile sense was unchanged, over the remainder of the fingers sensibility was not changed from what it was previous to the section of the roots. It is particularly noteworthy that the *palmar surface of fore and middle fingers*, a region generally given to the sixth root, was *not* anæsthetic.

The following conclusions as to the root supply, then, seem to be justifiable in this case:—

(1) The absolutely anæsthetic strip along the radial edge of the forearm corresponded with that ascribed by both Starr and Kocher to the fifth root and must have been due in part or wholly to the section of this root.[1]

(2) The sixth root in the hand could not have supplied more than the thumb (front and back) with its ball, though it probably aided in giving sensory fibres to the *back* of the forefinger (increased loss of pain sense). This is a much more limited area than that usually given to the sixth cervical,[2] as will be seen by comparison with diagram II.

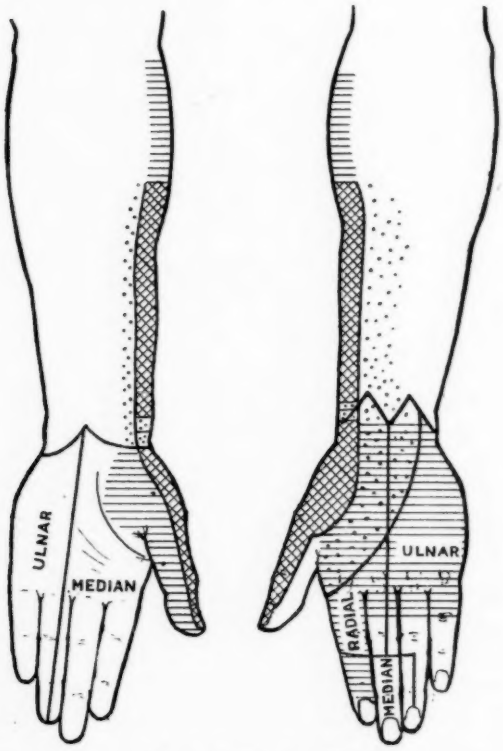
(3) The seventh cervical *probably* gave a more extensive supply to the back of the hand than usual, but not to the fingers. It may have contributed to the partially anæsthetic areas over the first phalanges of the fingers. The further distribution of the seventh, either to the palmar or the

[1] The section of the sixth root may have contributed to this area as given by Wichmann.

[2] As the seventh was cut, the preservation of sensation over the fore and middle fingers could not have been due to overlapping of this root.

dorsal surfaces, was not demonstrable. The area of impaired sensibility along the back of the forearm (dotted in the photograph) could not have been due to section of the seventh cervical, as it was present before the operation.

(4) The remainder of the backs of all four fingers and



DIAGRAMS III. AND IV.

Taken seven months after operation and showing the relation of the anæsthetic areas to the distribution of the ulnar, median and radial nerves; also restoration of sensibility. Cross shading = loss of pain and tactile sense; transverse lines = analgesia; dotted areas = diminished tactile sense (in forearm the limits were not determined at this date). Tactile sensibility had returned over ball and palmar surface of thumb and partially or wholly over a large part of back of hand. The analgesia is substantially that immediately following sections of the fifth, sixth and seventh cervical roots.

nearly the whole of the palm and palmar surfaces of the fingers must have received a supply from the eighth cervical or first dorsal, or both, which would thus have a wider distribution than that usually assigned to them.

The probable explanation of these apparent variations in the root areas is to be found in a corresponding variation in the termination of the ulnar and median nerves. The ulnar nerve is supposed to supply ordinarily the inner third of the palmar surface of the hand (diagram III.), but variations occur. This nerve derives its fibres from the eighth cervical and first dorsal roots, and if it had an unusually wide distribution in the hand, these roots would supply areas ordinarily given to the sixth and seventh cervical through the median; or possibly the eighth cervical root fibres contained in the median might be more widely distributed than usual, and some be continued into the branch of the median that supplies the area in question. The variation shown on the back of the hand is still more interesting. Here the fingers showed no increased anæsthesia, excepting near the proximal knuckles, and even here it was not absolute. Now the median (diagram IV.) supplies a quadrangle covering the distal half of the backs of the first three fingers approximately. It is presumable, therefore, that in this case the fibres which the median derives from the eighth cervical must have been distributed to this quadrangle, and not those from the sixth and seventh, which were cut, as would be the case according to the diagram of Starr and others; and further, that the median had a wider distribution over the whole of the backs of the fingers. A reference to diagram III. will make this clear.

Very remarkable was a later return of sensibility in previously anæsthetic areas. On April 10, about five months after the operation, it was noted that above the shoulder the anæsthetic area had retreated from the neck to the shoulder-joint, and corresponded with the circumflex nerve area (dotted line in the photograph); in the arm sensation had crept in to some extent on the lateral borders. In the hand, likewise, tactile sensibility was returning. Two months later still (June 22), a quite extensive return of the

tactile sense had occurred. This Jerry himself had noticed by testing himself.

He feels the lightest touch over the ball and palmar aspect of the thumb, though here there is still analgesia. Only over the dorsum of the thumb is there absolute loss of both pain and tactile sense. Over the dorsum of the hand he feels a very light touch to the ulnar side of a curved line shown in diagram III., but between this line and the area of absolute loss of sensation he feels a firm touch only. The analgesia remains as before. In the forearm there has also been considerable return of the tactile sense. As the roots were freely cut, it is difficult to suppose they could have re-united; the return of sensibility must therefore be due to overlapping nerve supply from adjoining root areas, which have become functionally more exciteable. It is not easy to explain the return of tactile sense, without improvement in the sensibility for pain. As to the Brown-Sequard paralysis, he complains bitterly of a burning feeling in the *right* thigh and coldness of the right foot; if cold water is dropped on any part of the leg he says it feels like a "hot iron." There is no sensory irritation on the left. The left knee-jerk is still lively, and, although there is no ankle clonus, there is a violent Babinski accompanied by a similar dorsal flexion of the foot and trepidation of foot and leg. In right foot there is a comparatively slight Babinski. The paresis of *left* leg is about the same as when last noted.

A CASE OF BILATERAL PORENCEPHALY.

BY J. WIGLESWORTH, M.D., F.R.C.P.LOND.

THE subject of this case was a female idiot, aged 24, who died in Rainhill Asylum on May 16, 1899, after a residence of nine years. Her parents were natives of Queenstown and each of them was 38 years of age at the time of marriage; they were steady sober people. A first cousin—the daughter of a maternal uncle—died of melancholia in Cork Asylum, but this was the only case of mental or nervous disease in the family so far as could be ascertained, though there had been several cases of phthisis. There were three children from the marriage, two girls and a boy, patient being the third child; the other two were all right mentally, but the eldest girl was not strong physically. All the confinements were easy ones, and there had been no miscarriages. Patient is described as having been always a backward child, and was late in walking and speaking. When a year old she had a bad fall down stairs, with, however (the father said), no visible ill effects. At the age of 3 she had a fit for the first time and continued to have these about every three months or so until she was five years old, when they ceased; the fits were severe and appear to have been of an ordinary epileptic character. During this period what intelligence she had gradually failed, and she does not appear to have improved with the cessation of the fits, for her condition is said to have remained very stationary from that time. A few months previous to admission she had what are described as attacks of "stupor." She began to menstruate when 13 years of age.

When admitted she was found to be a small undersized

girl, weighing only 6 stone 12 pounds. She had considerable paresis of the right arm and leg, but could walk, although with a shuffling gait, dragging her right foot after her, and she had a certain amount of power in the right arm, though she seldom used it. She had double strabismus. Mentally she was a decided idiot. She was usually very dull and lethargic, sitting about silent, listless and unoccupied. She never spoke spontaneously in an intelligent fashion, but when spoken to she repeated over and over again some of the words addressed to her in a parrot-like manner without attempting to reply; similarly she would sometimes shout out snatches of songs which she heard other patients singing, and she would often laugh and chatter to herself in a meaningless way. She was never vicious, impulsive or violent. She was at all times wet and dirty in her habits and required a good deal of attention, having to be washed and dressed; she fed herself, however, using both hands for this purpose. At times she had attacks of restless excitement, when she was noisy, destructive and degraded in her habits, plastering herself with her fæces. In spite, however, of her fatuous appearance she understood many simple things that were told her, and when at her best could be got to do a little simple work, such as carrying things about. She eventually died of phthisis.

AUTOPSY (*an examination of the head only could be obtained*).

Circumference of head 21½ inches.

Skull, slightly asymmetrical, the temporal and occipital fossæ being apparently larger on the right side than the left.

Encephalon (after draining off all fluid) weighed 852 grammes.

Left hemisphere (fig. 1).—Almost the whole of the outer aspect of the posterior two-fifths, or rather more, of this hemisphere, is converted into a large thin-walled cyst communicating freely with the left ventricle, and in fact forming with this one large cavity; the outer wall of this cyst is for the most part exceedingly thin, consisting merely of thickened pia-arachnoid and ependyma (probably) but in

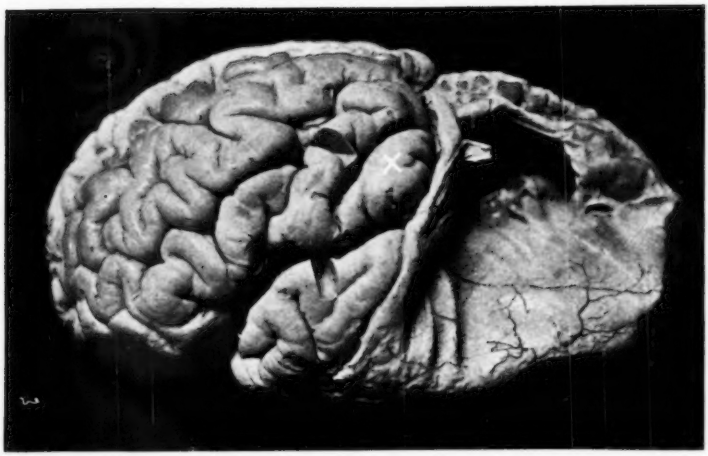


FIG. 1.

Left hemisphere. The × is placed on the lower part of the ascending parietal convolution.

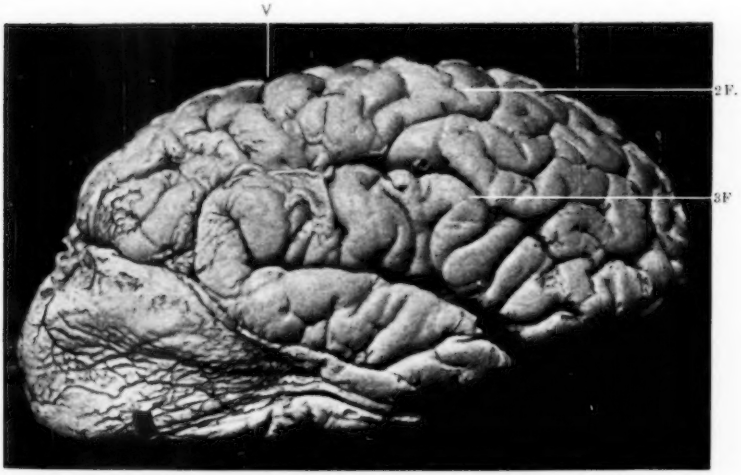


FIG. 2.

Right hemisphere. The arrow indicates the upper end of the fissure of Rolando.
2 F. — 2nd frontal convolution. 3 F. — 3rd frontal.

places, especially as the base of the cyst is approached, it is formed in part also of thinned out cerebral tissue. The cyst is bounded in front at the point where it abuts on the healthy brain tissue by a well-defined line, which is for the most part nearly vertical, cutting the fissure of Rolando across at about its middle, but curving slightly forward at its lower end where it runs into the temporo-sphenoidal lobe. Over the whole of this area the convolutions have entirely disappeared with the exception of a band of puckered and atrophied convolutions at the upper margin of the hemisphere, which appears to represent the remains of the paracentral and superior parietal lobules. The convolutions destroyed therefore comprise the upper third of the ascending frontal, the upper half of the ascending parietal, nearly the whole of the superior parietal lobule, and the external occipital convolutions, a small fragment of the posterior convolutions of the insula, the whole of the supra-marginal and angular gyri, and the posterior half or rather more of the first and second temporo-sphenoidal convolutions, whilst the anterior half of this latter gyrus, together with the whole of the third temporo-sphenoidal, are greatly thinned; on the basal aspect the convolutions entering into the fusiform and lingual lobules (forming the floor of the cyst) are also greatly atrophied, though still showing some remains of their original division; the hippocampal and uncinate gyri are, however, intact, as also is the anterior half (about) of the first temporo-sphenoidal gyrus. The whole of the convolutions on the outer aspect of the hemisphere, in front of the vertical line above mentioned, including the convolutions of the orbital surface, the anterior half of the first temporo-sphenoidal, and almost the whole of the insula, are intact and well developed; whilst on the inner aspect of the hemisphere the convolutions for about the anterior two-thirds are also well developed; but those comprising the quadrate lobule, and more especially those of the cuneus, are distinctly thinned and have lost their normal arrangement, running from above downwards in more or less of a radiating fashion, those of the cuneus indeed being so thinned out as to be undecipherable.

Right hemisphere (fig. 2).—This shows a somewhat similar lesion to that in the left hemisphere, but the destruction of brain tissue is here much less, and the cyst which is formed by a hollowing out of the occipital and temporo-sphenoidal lobes is correspondingly smaller, although forming with the dilated posterior part of the lateral ventricle a cavity of considerable size. The wall of the cyst, too, is not so thin as is that in the left hemisphere, consisting of a layer of thinned-out brain tissue, together with pia-arachnoid, and being nowhere formed by membrane only; at the tip of the occipital lobe, however, where the wall is thinnest, it is barely 1 mm. in thickness. The stress of the lesion has fallen on the convolutions of the occipital lobe, the posterior part of the parietal lobe (more especially the angular gyrus) and part of the posterior portion of the temporo-sphenoidal lobe, the part of this lobe which has suffered most being the area comprised in the second temporo-sphenoidal convolution, which is extensively destroyed, forming a tongue-shaped band of atrophied brain tissue running forwards between the first and third temporo-sphenoidal convolutions, the former of which is intact and the latter fairly so, especially in the anterior half, but it thins off considerably towards its posterior extremity where it forms part of the lower and outer wall of the cyst. Over the area just named which forms the outer wall of the cyst, the convolutions are quite undecipherable, the brain tissue which remains consisting merely of a thin smoothed-out layer varying from 1 to 3 mm. in thickness. Interiorly the convolutions of the cuneus are also much puckered and atrophied, and those comprising the fusiform and lingual lobules, forming the floor of the cyst, are also atrophied, although to a less extent; the hippocampal and uncinate gyri appear normal as in the opposite hemisphere. In front of the area just described as forming the main seat of the lesion, the convolutions of the parietal lobe are found to be much puckered and contracted, and they have lost their normal arrangement, those which have suffered least being the upper two-thirds of the ascending parietal and the superior parietal lobule. The first temporo-sphenoidal gyrus is intact and the supra-marginal gyrus, though small, is also distinct. An-

teriorly to the fissure of Rolando the convolutions of the outer aspect of the hemisphere are all plump and well-formed. The convolutions of the frontal lobe however, though well developed, are abnormal in their distribution since they run back in parallel lines right to the fissure of Rolando, and the ascending frontal convolution being thus broken up by the sulci which join this fissure, cannot be distinguished as a distinct and separate convolution throughout its length; there is, moreover, no distinct præcentral sulcus. The orbital convolutions are normal, as are also those of the insula. On the inner aspect of the hemisphere, the convolutions are well developed as far back as the parieto-occipital fissure, and present a fairly normal arrangement except that the convolutions of the quadrate lobule run in parallel lines towards the posterior part of the corpus callosum.

Pons presented no marked asymmetry to the naked eye, but on microscopical examination the fibres of the pyramidal tract on the left side are found to be distinctly smaller than those on the right.

Medulla shows pronounced asymmetry, the pyramid on the left side above the decussation being about half the size of the right one.

Cerebellum, normal; no marked asymmetry.

Microscopical Examination of Cerebral Cortex (made by Dr. A. W. Campbell).

Sections were taken from about the middle of the following convolutions, the method of Nissl being employed for displaying the nerve-cells, and the nerve-fibres being dyed with hæmatoxylin, after fixing with osmic acid or iron alum.

Frontal second. Right.—Depth of cortex, 2·941 mm.; the reduction of depth is uniform, there being no striking wasting of any particular layer. The nerve-cells appear numerically deficient, but not to a pronounced degree, and the layers are arranged in a normal manner. In cells of sufficient size to allow of an examination of the arrangement

of the chromophilic particles (larger pyramidal cells), it is found that these elements, though present, stain indistinctly. There is a perceptible reduction in the nerve-fibres in all parts; the tangential band consists only of about half a dozen fibres: the supra- and intraradiary horizontal fibres (Baillarger's lines) are poorly represented, and the vertical striæ of Meynert are thin, and also widely set apart.

Ascending frontal. Left.—Cortex at summit, 3·383 mm. in depth. The nerve-cells are normally arranged, but again deficient in number. The large pyramidal cells show a normal arrangement of chromophilic particles. The tangential band of nerve-fibres is poorly represented, but otherwise the cortex is well supplied with fibres.

Paracentral lobule. Right.—Cortex at summit, 2·55 mm. in depth. The nerve-cells and nerve-fibres are both somewhat deficient. The large multipolar nerve-cells appear to be normal.

Temporal first. Left.—Depth of cortex at summit of convolution, 3·40 mm. Nerve-cells again deficient in number. There is a good tangential band of fibres, but the horizontally directed supra- and intraradiary fibres are not present in normal abundance, and Meynert's striæ are thin.

Temporal third. Right.—These sections are taken from an area where the cortex and white matter together measure only 2·397 mm. in depth; of this, 1·154 mm. represent cortex, the rest white substance and ependyma. In spite of the extreme thinning of the cortex, the nerve-cells, although diminished in number, are yet present in considerable abundance, and their disposition in layers is not altogether destroyed; but they are all of diminutive size and mostly of pear shape, with an apical process only. The body of the cell is mainly occupied by nucleus, and chromophilic particles are not recognisable in the cell-body. In the position normally occupied by the tangential band there is a dense feltwork of nerve-fibres interlacing in all directions. Elsewhere there are few horizontally directed fibres, but there are many vertical ones; these, however, run singly, and are not collected into bundles, as they are in the normal radiations of Meynert.

Remarks.

Cases in which porencephalic defects occur on both sides of the brain, though less numerous than unilateral ones, are nevertheless not altogether uncommon ; thus Audry found the lesion to have existed on both sides in one-third of his collected cases [1] (thirty-two out of ninety-six). That the loss of cerebral substance in this case was due, not to a developmental defect, but to a definite lesion, appears to be clear, though it is less easy to feel sure as to the exact nature of the lesion. There can be little doubt, however, that it occurred either during intrauterine life or in early infancy. There seems to have been nothing in the actual process of birth to have favoured a traumatic origin, and though there is a history of a somewhat severe fall down stairs when the patient was a year old, we have the definite statement of the patient's father that this fall produced no visible ill effects, and therefore it is difficult to associate the lesions with an injury at that period, even though these occurred in parts of the brain which would perhaps least readily give rise to symptoms in an infant of that age. On the whole it seems probable that the cerebral defects in this case owe their origin to obstructive vascular lesions occurring in intrauterine life. Though the area of brain destroyed does not follow with complete exactness the usual lines of arterial distribution, there is, of course, considerable variation in the vascular distribution in different brains, and it may be said generally that those portions of the brain supplied by the anterior cerebral arteries and the anterior branches of the middle cerebral have escaped, whilst the stress of the loss has fallen on the parts to which the posterior cerebrals and the posterior branches of the middle cerebral are distributed. The lesion in the right hemisphere differs from that in the left hemisphere chiefly in the facts that it occupies a smaller area and that the destruction of the brain tissue involved has not progressed to the same extent as in the left. In no part of the area involved in this (right) hemisphere does the

[1] "Les Porencéphalies," *Revue de Médicine*, June and July, 1888.

brain tissue seem to have disappeared entirely, although in places it is so very greatly reduced in thickness; and this fact favours the idea that the process has, in part at any rate, been a gradual atrophic change due to a slowly-progressive or partial obliteration of vessels, rather than to a single sudden lesion, which would have totally destroyed the brain tissue over a limited area. In the left hemisphere, however, the cerebral substance has gone altogether over a considerable area, and the completeness of disappearance very probably stands in relation to the greater activity of the absorptive process in these early cases, as compared with what obtains in later life. The fact that there was an appreciable deficiency in the number of nerve-cells and fibres, even in those parts of the cortex which appeared normal to the naked eye, is an interesting point, but one which might have been anticipated, as with so considerable an area of the brain practically non-existent, it could not be supposed that the portions which remained could develop the same wealth of connections as in a normal brain.

FRIEDREICH'S PARALYSIS.

BY T. DUNCAN GREENLEES, M.B.EDIN., F.R.S.E.
AND
G. CARRINGTON PURVIS, M.D.EDIN., B.SC.

FRIEDREICH'S ataxy is one of those forms of chronic spinal paralyses, standing midway between disseminated sclerosis and locomotor ataxy, and which is considered a comparatively rare disease.

The cases here described are, so far as we can ascertain, the first published in South Africa, and indicate, as has been previously pointed out, that the European inhabitants of this country are rather prone to degenerate nerve lesions.

The following cases occurred under our care at the Chronic Sick Hospital, Grahamstown, in a boy and girl—brother and sister. The girl died last May, and we were fortunate in securing a post-mortem examination. The boy still lives, and a careful clinical examination has been made of him. We are thus able to present a double picture—of the clinical symptoms, as found in the boy, and a microscopic description of the lesions, as found in the case of the girl. The symptoms, in both cases, were so nearly identical that we think the following description can be easily made to apply to both, and that, whereas we regret being unable to give the clinical picture history of the girl, we are at the same time in a similar position as regards the pathological details which may be found in the case of the boy.

CLINICAL DESCRIPTION.

Family history.—Careful enquiries were made to ascertain the family history of our patients, from their mother,

with the special object of ascertaining whether there existed a hereditary tendency to nerve troubles. The parents are not blood-related to each other, as so often occurs amongst the Dutch of this country; they are reported both to be healthy, and the father is, or was, fighting for the Transvaal. They had nine children, all of whom were in good health, except our patients, and one child who died from "convulsions" in infancy. The father is reported to be steady and temperate in his habits, is a poor farmer, and lives a healthy out-door life. The mother is in good health and of average intelligence. There are no known cases of paralysis in the family.

Onset of illness.—When Charlie was 9, and Nellie 12 years of age, they both had scarlatina, and while convalescing they got wet one day. It was noticed, shortly afterwards, that both children were weak in their ankles, and gradually the feet began to contract so as to make walking well nigh impossible. The disease progressed very slowly at first, but the parents noticed that both children were steadily getting worse. The contraction of the feet increased, and paralysis gradually affected both legs and arms so that the movements of the limbs became more and more limited in their range, and they became very helpless accordingly. They were admitted from Kimberley on March 11, 1896, being then 13 and 16 years of age respectively.

At that time the girl was very helpless indeed; she was unable to assist herself in anything, and had to be dressed and undressed and fed by the nurse. She sat on a chair all day with her head resting backwards on a pillow; gradually the general feebleness increased; she had several fainting attacks, and during the last few months of her life she had difficulty in swallowing and the respiratory functions became interfered with without pulmonary disease at all. The dysphagia was so distressing that, towards the end of her life, she was quite unable to take nourishment by the mouth.

That the disease, in her case, was one which ultimately implicated the higher nerve centres was evident from the symptoms which, in the end, proved fatal. The difficulty in

Fig 1

FIG. 2.

FIG. 3.

breathing, the fainting attacks, and the dysphagia, all indicated some lesion affecting the organic centre in the medulla, and this idea was borne out by the microscopic examination of this portion of the nervous system.

Clinical examination of Charlie.

Charlie is a boy now (October, 1900) aged 17 years. The expression of his face is that of happiness and simplicity combined—almost amounting to imbecility. His face is broad and flat, he always wears a childish smile, and he never loses his temper, nor looks cross. He is quite unable to articulate, but he knows what is said to him. His conduct generally is good, he is very patient, and a favourite with the other imates of the hospital, who are always ready to assist the helpless boy. As is general with cases of slight mental defect, he is somewhat vain, and likes to wear cheap medals, brass chains, and rings on his fingers.

He is utterly helpless, is unable to walk, and, when moved or carried, his head falls limply from side to side as if the neck muscles were unable to support it. While there is a general wasting of the muscles there is no marked impoverishment of nutrition, and his appetite is very good.

His legs hang helplessly down, and when moved about he grips tight hold of the arms of the chair lest he should fall. When at rest both feet are slightly turned inwards, and there is well marked talipes equinus of both feet. This condition is more evident in the right foot, as also is a turning inwards of the foot so that the sole faces almost upwards—approaching to the condition of talipes varus. The great toes of both feet are hyper-extended, a condition likewise most marked in the right side (figs. 1 and 3).

The Spinal Column.—An examination of the back reveals marked lateral curvature, the convexity occurring in the upper dorsal region, and points towards the left side. The apex of the curvature approaches very closely to the posterior angle of the scapula (fig. 2).

Electric Reactions.

With galvanism:

(1) *Flexors of forearms.*

20 cells: Ma = 3.
KCC +; ACC +; AOC 0; KOC 0.
Note.—KCC slightly stronger than ACC.

27 cells: Ma = 4·3.
KCC +; ACC +; AOC 0; KOC 0.

27 cells: Ma = 4·75.
KCC + strongly marked; ACC + strongly marked;
AOC + very slightly marked; KOC 0.

32 cells; Ma = 10·5.
KCC + strongly marked; ACC + strongly marked;
AOC + slightly marked; KOC + very slightly marked.

(2) *Leg muscles* (posterior and outer aspects).

20 cells: Ma = 2.
KCC +; ACC 0; AOC 0; KOC 0.

25 cells: Ma = 3.
KCC +; ACC +; AOC 0; KOC 0.

30 cells: Ma = 6·5.
KCC +; ACC +; AOC 0; KOC 0;

35 cells: Ma = 10.
KCC +; ACC +; AOC 0; KOC 0.

It will be seen that the electrical examination—so far as it goes—presents reactions which are compatible, or nearly so, with health.

On applying the *Faradic current* it was noted that the muscles of both arms and legs were slow in responding to the interrupted current; as regards the legs a fairly strong current was required to make the muscles contract.

Reflexes.

(1) *Superficial.*—The plantar, cremasteric and abdominal superficial reflexes were tested and were only slightly

FIG. 4.

FIG. 5.

FIG. 6.

evident, while the epigastric, gluteal and interscapular reflexes could not be elicited.

(2) *Deep.*—Both the patellar tendon reflex and the ankle clonus were absent.

(3) *Organic.*—All the organic reflexes were normal except the act of micturition which was somewhat delayed.

The hot sponge test.—The patient felt it hot in the lumbar region as well as in the dorsal region, but only when first applied. The test failed on repetition.

Hot tube test.—Was not felt when applied to either the legs or the arms.

Pain sensation.—The patient felt a needle-prick in various parts of the body, but the needle had to be firmly pressed into the skin.

Muscular sense.—He was unable to differentiate between weights. When asked to touch the tip of his nose rapidly with his finger, he failed to do so, but when allowed to take his own time the act was slowly performed, his hand swaying from side to side while his finger approached his nose.

When told to hold out his hands he presented distinct drop wrist, but when he is asked to straighten them out the fingers are extended backwards to an extraordinary extent. This hyper-extension of the fingers is most marked in the right hand. There is wasting and probably some paresis of the interossei muscles of both hands (figs. 4, 5 and 6).

On being asked to put out his tongue he does so in the middle line; the tongue is largé, flabby, and there are general tremulous movements of the organ.

His pupils react readily to light and accommodation, and there is slight nystagmus of both eyes.[1]

The other senses seem perfectly normal, and he complains of no objective symptoms such as pains in the head or sounds in his ears.

Pathology of Case of Friedreich's Disease.

The following remarks are founded upon the *post-mortem* examination of Nellie B., the sister of the above patient,

[1] Nystagmus was a marked symptom in patient's sister.

who died at the Chronic Sick Hospital on May 10, 1900, after a residence of four years, and in whom the disease had existed for nine years.

Autopsy (twelve hours after death).—Body of a female European girl, much emaciated and muscles wasted. Rigor mortis present. Double talipes equino-varus marked. Small bedsore over sacrum. There was some wasting of the cerebral convolutions, with an excess of fluid in the cranial cavity. The veins of the pia were congested.

The heart and lungs were healthy except for some congestion of the lower lobe of the right lung, and the bronchial tubes contained frothy material.

With the exception of a small cyst in the centre of the left kidney, the abdominal organs presented nothing calling for special note.

A piece of the left frontal convolution, the crus cerebri, medulla oblongata, a portion of the great sciatic nerve, and portions of the cervical, dorsal, and lumbar regions of the spinal cord were placed in Müller's fluid, and forwarded to the Bacteriological Institute, Grahamstown. These were hardened and stained according to Weigert's method, and microscopic sections made.

Examination of the Spinal Cord.

An examination of sections from different portions of the spinal cord revealed the fact, which was even quite apparent to a naked-eye inspection, that the cord was much smaller than it should be. It was quite infantile in size, and it looked even smaller than a healthy cat's cord, with which it was compared.

Microscopical Examination.

Sections from various levels in the cervical, dorsal, and lumbar regions were examined microscopically, and revealed the following pathological changes :—

Cervical region at the level of the sixth cervical vertebra :—

(1) Sclerosis of the middle portion of the posterior internal tract (Goll's tract).

FIG. 7.

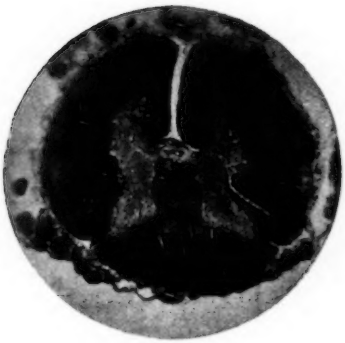


FIG. 8.

FIG. 9.

(2) Sclerosis of the posterior external tract (Burdach's tract) external to the comma tract.

(3) Sclerosis of the ascending antero-lateral tract (Gowers' tract). This occurs as a small patch of sclerosis just where the anterior nerve-roots leave the cord.

(4) Sclerosis of the direct cerebellar (lateral) tract. This occurs on both sides of the cord to a marked extent, nearly all the nerve-fibres being replaced by fibrous tissue.

(5) In Clarke's column the cells were few in number, markedly atrophied, took the stain badly, and the intercellular tissue was much broken down.

(6) Lissauer's tract, with the exception of a small portion adjoining the direct cerebellar tract, was healthy, as also was the crossed pyramidal tract.

Dorsal region at the level of the second dorsal vertebra :—

The lesions described as occurring in the cervical region were evident, but not to so marked an extent. The sclerosis of the posterior portion of the direct cerebellar (lateral) tract was, however, even more marked, and occurred as a distinct patch, under a low power, of lighter hue than the surrounding healthy nerve-fibres, and this condition was equally marked on both sides of the section (fig. 7).

Clarke's column was affected to a similar extent as existed in the cervical region ; its cells and fibres were both affected extensively.

Lumbar region at the level of the third lumbar vertebra :—

In this portion of the cord the sclerosis was very evident, and quite localised to a patch on either side of the posterior median fissure, embracing Bruce's and the comma tract, but leaving the free edge of the fissure healthy. A small patch of sclerosis likewise existed on either side of the posterior fissure more centrally situated and close to the commissure.

The degenerative changes in the lateral and anterior columns, although present, were not so evident as they were in the cervical region of the cord (figs. 8 and 9).

When a higher power was employed it was noted that, in some parts of the diseased portions of the cord, the nerve sheath appeared simply empty of its axis-cylinders. In other portions the axis was either shrivelled or else swollen.

As a result of the former condition, a honeycomb-like arrangement existed, consisting of empty nerve sheaths closely packed together, but where the disease appeared more advanced even the nerve sheaths became obliterated, and replaced by fibrous tissue, so broken down in places as to leave large, almost empty spaces.

The nerve-cells, for the most part, stand out prominently in all the sections; indeed, this prominence is unusually marked where it does exist. In the dorsal region, while the cells of one anterior horn were sharply defined and of a deep orange colour, those in the other horn were hardly evident at all and of a faint primrose colour, and were apparently atrophied, their dendrites and axons being shrivelled. Whereas in the former horn we counted, in one section, six large well-defined cells, the corresponding horn on the other side only showed two pale yellow cells.

The horn in which these faintly-stained cells were found was perceptibly smaller than the opposite anterior horn.

A further examination of these cells revealed several interesting points in cellular pathology. The faintly outlined cells appeared to be swollen, their nuclei indistinct and in some cells pushed out so as to form a prominence in the contour of the cell, and the nucleolus alone appeared sharply defined as a shining point. The contents of the cells were granular and there was an entire absence of Nissl-bodies or any attempt at an intracellular structure.

The cells, on the other hand, which presented a prominent appearance in the various sections, differed entirely in every respect from the others. They were sharply defined, of a darker orange colour, and in contour looked like orange-coloured black berries. They were more rounded than exists in health and their sharp edges had disappeared. In few examples were either axon or dendrite apparent. Nissl-bodies were absent, and chromatolysis in most of the cells was complete. Their contour was so peculiar and unusual that in no drawings illustrative of the pathological changes occurring in nerve-cells, given by Ford Robertson in BRAIN, 1899, and Ewing in *Archives of Neurology and Psycho-Pathology*, Vol. I., No. 3, have we discovered anything resembling the changes herein met with.

We can hardly consider this cellular change—mulberry-like alteration in its contour—to be due merely to "yellow globular degeneration," which, according to Colucci, is due to a morbid metabolism in the cell contents. The change is a change in the general shape of the cell whatever its cause may be, and is not altogether, at all events, the same pigmentary degeneration as that which is quite consistent with health.

Lugaro maintains, and perhaps rightly, that the displacement of the nucleus, as if it were pushed out from the body of the cell, bulging its contour in one place, is due to destruction of the chromatic reticulum which normally holds the nucleus in its central position, and Schäfer holds that the degeneration of nerve-cells in this, and similar spinal diseases, is due to degeneration of the posterior roots and consequent loss of the stimuli normally received therefrom. In fact, that loss of function first produces atrophy of the cell and ultimately degeneration of its contents.

The blood-vessels, as seen in the various sections, were full of blood, and their coats did not appear to be thickened to any extent.

The *Canalis centralis* was somewhat larger than normal, its lining epithelium appeared distinctly and was apparently quite normal.

The *Cerebral Cortex* (left frontal convolution) and the *Crus Cerebri* appeared quite healthy.

The *Medulla Oblongata* presented the following pathological changes:—

(1) The fasciculus gracilis (Goll's tract) was generally sclerosed.

(2) Sclerosis affected the restiform body, and the cells were only faintly stained.

(3) Gowers' tract took on the stain badly and the axis cylinders appeared to be atrophied.

(4) The fillet fibres were normal anteriorly, but posteriorly there was general sclerosis with isolated patches of nerve-fibres which were undoubtedly degenerated.

The Sciatic nerve in cross sections presented changes which were markedly pathological. The nerve-fibres were

closely packed together and pressed into masses by an increased deposit of fibrous tissue, which in places was broken down into debris-like accumulations in which no nerve fibres were observed at all. Some fibres were devoid of axis cylinders altogether; in others the axis was swollen. The empty appearance presented by the sheaths in places was very characteristic and looked like a honeycomb arrangement.

There was no uniformity in the size of the nerve-fibres; some were large and some were small. Whether the latter were due to simple pressure or whether they are actually embryonal structures is doubtful.

In sections stained with hæmatoxylin (Weigert) there were patches of the section which failed to take on the stain at all, consisting of very small nerve-fibrils, much atrophied and evidently much compressed by replacing fibrous tissue. These patches of sclerosis were all the more evident for the other portions of the section took on the stain admirably.

Remarks.

In Brain (1898, page 72) there is an interesting clinical record of several cases of Friedreich's ataxy by Dr. Mackie Whyte, and in the same volume at page 438 there is a very thorough article on the pathology of this disease by Dr. H. Mackay—both papers being supplemented by an extensive bibliography.

It may here be of interest to review the symptoms presented by our case which justified our diagnosis.

(1) Two cases occurred in the same family. The occurrence of paralysis, of a progressive type, in more than one member of a family during childhood should make us suspect this disease, and lead us to further observation.

(2) The disease commenced in both our cases immediately after a post-scarlatinal chill, and most observers hold that, as an exciting factor, some acute disease, especially if accompanied by fever, is generally the direct cause of the paralysis, the predisposing cause, of course, being a tendency to degeneration of the neuron.

(3) The first symptom noticed in our cases was a difficulty in walking—the ataxia being progressive in character.

(4) There was a rapid change in the power of articulation, so that, early in the history of the disease, the power of speech became well nigh abolished.

(5) As a later symptom in the progress of the disease, ataxia of the hands occurred; and hyper-extension of the fingers and toes, especially of the right hand and foot, is a prominent symptom in the case of Charlie.

Main en griffe—claw-like hand—is a condition which all along has been absent in our cases, although it is described as occurring in several of Dr. Mackie Whyte's patients.

(6) Athetoid movements of the fingers and toes, especially when the attention is diverted, were well marked in the case of the girl, although they do not form so prominent a symptom in the boy.

(7) An utter helplessness of the body exists in the case of the boy, and was very marked in the girl. The patient has to be carried about from place to place; he is quite unable to support himself unless when he is seated, and then he lies back on the chair, or else when he is lying down in bed. His head falls from side to side as if the neck muscles were quite unable to support it.

(8) There is marked dorsal spinal curvature—lateral; the apex of the deformed spinal column reaching the posterior border of the scapula in the case of the boy, and scoliosis was just as marked and in the same position in the girl.

(9) Talipes equino-varus, especially of the right foot, occurs in the case of the boy. Double talipes equino-varus existed in the case of the girl.

(10) Nystagmus was a very marked symptom in the girl. It is only slightly evident in the boy.

(11) The visceral reflexes in micturition and deglutition were affected; the former slightly in the boy's case; the latter, to a marked degree, towards the end of the girl's life.

(12) There existed marked incoordination in both cases. They were unable to touch the point of the nose without

first some hovering and hesitation of the finger in its progress.

(13) Electrical reactions in the case of the boy, so far as they go, were practically normal.

(14) There was an entire absence of the knee-jerk, and most of the deep reflexes were either impaired or abolished altogether.

(15) Finally, the intellectual faculties in both cases were only slightly impaired; no more, indeed, than might be expected in any case of chronic paralysis.

One curious fact about one case is that although the motor symptoms are mostly bilateral, still the ataxia seems more marked on the right side than on the left, and it may be that the spinal curvature pointing to the left side is due to the more advanced paresis of the right arm as compared to the left. This explanation is given by Mackie Whyte.

As regards the differential diagnosis of Friedreich's paralysis it is to be noted that there is a close similarity between it and hereditary cerebellar ataxia. Marie's theory, as pointed out by Whyte, may be correct, that the same degenerative process affects the spinal cord in the one case and the cerebellum in the other.

From disseminated sclerosis and locomotor ataxy, Friedreich's disease may be distinguished clinically by its family character, its occurrence in childhood, and an absence of many of the symptoms characteristic of these diseases, such as jerky "intention" tremors and ocular affections in disseminated sclerosis, and the "girdle" pain, and various crises—gastric and otherwise—of locomotor ataxy.

It should not be forgotten that cases have been described, among other observers by Friedreich himself, in which the symptoms indicated disease of a hybrid nature. Such cases are, however, the exception, and do not disprove the fact there is a group of symptoms, affecting the motor nervous system and characterised by distinct lesions in the spinal cord, medulla and nerves, forming a distinct entity and to which Friedreich gave his name.

A consideration of the pathogenesis of Friedreich's disease is both interesting and instructive. Dr. Pitt (*Guy's Hospital*

Report, 1886-7) puts forward the theory that this disease, with its imperfect development of the spinal cord, is vascular in its origin, because—

(1) The posterior columns of the cord are the most vascular, and here the sclerosis is most marked.

(2) Vascular lesions have been described in some cases, and the disease is always aggravated by acute affections.

(3) Development of the cord begins in the most vascular regions, so that degeneration attacks, by preference, the latest developed parts.

(4) The sclerosed regions correspond to tracts, the filaments of which are covered by myeline after the fifth month of fœtal life.

(5) The symptoms become more marked at puberty, which is accompanied, as we know, by great vascular modifications in the organism.

And, again, Guizetti (*Reforma Medica*, June, 1893) maintains that Friedreich's disease "depends upon a congenital predisposition, in consequence of which, in the early years of life, certain systems of fibres and nerve-cells undergo a process of progressive atrophy, and that the process is independent of any contributory effect from vascular alterations."

Mackay describes these "certain systems of fibres and cells" quoted by Guizetti, as those which are latent to undergo medullation and those which are functionally related to the postero-internal tract.

The entire absence of disease of the coats of vessels in our case, although they were mostly full of blood, disproves somewhat Pitt's theory as to a vascular origin. On the other hand, while there may be a predisposing factor in developmental arrest and congenital malformation of the spinal cord, it is interesting to note that, in the majority of cases recorded, the first symptoms of the disease observed have followed upon some acute febrile state of the system, in which it is reasonable to suppose a congested condition of the spinal cord vessels existed.

We are disposed to favour both theories, in part. That the disease is due to an arrest in the development of certain strands of nerve-fibres and cells in the cord, the results of

which are not evident until some intercurrent acute affection occurs, sums up briefly our present ideas of the pathogenesis of Friedreich's paralysis.

It remains for us briefly to discuss the prognosis and treatment. As regards the former, Friedreich's ataxia is progressive and chronic, many cases living, according to Ladame (BRAIN, 1890, p. 525), to upwards of 40 years of age.

Although intermissions are said to have occurred, yet in no case has a recovery been reported. The disease either kills by itself or some intercurrent affection may cause a fatal termination.

As in all spinal cord affections, the duration of life depends, to a marked extent, upon the character of the nursing, and it is interesting to note here the almost entire absence of any tendency to bedsores occurring. Our patient sits day after day on the same chair, and yet his skin remains unbroken, and the girl's slight bedsore was due more to emaciation than to any trophic affection.

With such a prognosis little need be said as to the treatment of Friedreich's ataxy. Charcot recommended "suspension" with apparent benefit; Zalludowski gave temporary relief to a little girl by means of massage to the limbs, and Mackie Whyte recommends exercise and an education of the functions of those muscles which tend to become affected. Our patient says that he feels very much better after the use of the continuous current. Drugs seem to have little influence in the arrest of symptoms, and the most we can do for cases is to nurse them carefully and attempt to combat the graver symptoms as they occur.

In conclusion, we would express our thanks to Dr. le Doux, of the Bacteriological Institute, for his kindness in making sections and photomicrographs of various parts of the nervous system which illustrate this paper.

A CASE OF SYPHILITIC SPINAL LEPTO-MENINGITIS WITH COMBINED SCLEROSIS.

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THE patient in question first came under observation in the Royal Infirmary, Dundee, where he was under the care of Dr. Mackie Whyte, and afterwards in the Dundee Poorhouse Hospital, again under the care of Dr. Whyte, to whom I am indebted for permission to publish the notes of the case and for the material for microscopic examination. Altogether the patient was under observation from October, 1897, until February, 1900, when he died. The notes of his case while in the Royal Infirmary I give verbatim, as nothing of them is superfluous.

R. L., aged 61, labourer, was admitted to the Royal Infirmary, Dundee, on October 7, 1897.

Complaint.—(1) Pain in right side of chest; (2) shooting pains in left arm and leg.

Duration.—(1) One week; (2) two years.

Previous history.—He has always been a strong healthy man, never having had any illness, except measles or scarlet fever. His work has been heavy, and has necessitated constant exposure to weather. He has suffered from "rheumatism" at times, but has not been subject to chest troubles. He has been a total abstainer for eight or nine months, but before that was a heavy drinker. He is a non-smoker. There is no history of venereal disease. Micturition has been more frequent for the last nine months, obliging him to get up three or four times at night.

Family history.—Negative. Married. Wife and six children healthy.

Present illness.—Two years ago, for no reason, a pain came on one night in his left heel; the foot and leg as far as the knee

were swollen next day, but the swelling subsided without treatment and has never returned. The pains have continued more or less ever since, shooting sometimes from the heel up the leg, and up to the shoulder and down the arm, and sometimes in the reverse direction. Rubbing relieves the pain, which is severe enough to keep him awake at night. They have never affected the right side. In the left hand he often feels a sensation "like pins and needles." Walking is affected, as he never feels as if he were gripping the ground with his left foot. His memory has been failing for four or five years. The grasp of the left hand is weakened. A week ago a pain started in the right side of the chest, associated with a cough, worse at night, but relieved if he lies on his right side. He has lost three stone weight in four years. Suffers from frontal headache.

Physical examination.—He is a powerfully-built man of fairly good colour. Tongue clean and moist, not tremulous. Heart and lungs appear healthy.

There is tenderness all over the right side of the chest, extending as high as the third intercostal space, reaching to the middle line back and front, worst over the points of each of the intercostal nerves. There is tenderness in the right lumbar region and over the liver. No enlargement of liver or spleen. There is considerable loss of power in the left leg. Patellar and plantar reflexes exaggerated; no ankle clonus. Hyperæsthesia all down left leg, tenderness especially marked over the course of the sciatic nerve. No wasting or coldness, no ataxia; but the left leg is dragged when he walks, and he seems afraid to trust it. He says there is pain in the heel when walking, also that the ground is "not gripped properly." He can stand with his eyes shut, but cannot walk along a straight line. The grip of the left hand is very feeble. Dynamometer, right 30, left 10. There is hyperæsthesia of the left arm, and a feeling of pins and needles, especially in the hand. The pupils respond to light.

October 8. Urine: specific gravity 1006, acid, colourless, no albumen. The excess of sensitiveness is noticed on the scalp and face, as well as on the body, and is more marked on left side.

October 11. Sleeps well. Pain still severe in left side.

October 19. Patient exhibits a general condition of muscular weakness all over his body. His gait is tottering and shuffling, with very short steps, hardly raising his feet, but sliding them along. The left leg is distinctly the weaker. The arms are also very weak, the left more so. Dynamometer, right 30, left 20. He cannot protrude his tongue much further forward than his teeth. The movements of the eyes are good and the reactions of

the pupils normal, but the pupils are small, and the right somewhat smaller than the left. There is a general cutaneous hyperæsthesia all over the body from head to foot, and this is more marked on the left side than on the right. There is ataxia of the upper extremities, possibly due to weakness. No ataxia of lower extremities can be demonstrated, and the positions of the legs seem fairly well appreciated. Besides the hyperæsthesia, no affection of sensation is to be found. The deep reflexes appear exaggerated, possibly due to hyperæsthesia, as on the left side a tap on the knee or wrist evokes an exclamation of pain, and sets the muscles into spasmodic clonic contractions. No ankle clonus. Plantar reflexes well marked. Abdominal reflex well marked on right side. Takes food well, but has great difficulty in feeding himself. Peculiar odour about patient's skin. Sphincters act efficiently.

November 1. Cannot open mouth above three-quarters of an inch. Gums soft, and bleed easily. Speech is wanting in force, and such as one would expect from the weakened muscular condition.

November 19. Sent to poorhouse.

His temperature was usually slightly above normal, sometimes reaching 100° and once 101°. Towards the end of his stay it became subnormal.

From the notes of his case while in the Poorhouse Hospital I extract the following :—

November 7, 1898. Temperature 97·4°. Urine: specific gravity 1026, acid, no albumen. Patient was in hospital ten days ago for pain in, and inability to move, his right knee. He was put under chloroform, but nothing could be made out; the case was supposed to be one of hysteria. He now returns, complaining of pain in, and inability to move, the right hip-joint. On examination, nothing is made out.

May 17, 1899. Continues in a very helpless state, rigidity being the chief characteristic. This is especially to be observed along the spinal column, right shoulder, right hip, knee, and to a less extent in the right elbow and left knee. No joint deformity can be made out, and there is no special muscular atrophy connected with any joint, although the whole muscular system is below par. Attempts at movement cause pain. He manages to feed himself in a slow and tremulous fashion with his right arm.

January 19, 1900. Rigidity increasing. The left elbow and shoulder are now both involved, and the jaw is stiff. Pain only present on movement.

February 10, 1900. The patient can now hardly speak; his lower jaw is rigid, and the left arm is kept close to the side. The shoulder is also rigid, movements in the elbow are slight, the right fingers show voluntary movement. The pectoral muscles are firmly contracted, the muscles of the abdomen rigid and board-like. Lumbar region is arched. Breathing entirely abdominal and shallow. Head is rigid, somewhat extended, and rotation is impossible. Temperature in rectum usually 95·8°, never more than 96°. Patient has not spoken for two days.

February 16, 1900. Eyes strongly deflected to right, pupils small, and the head is more retracted. He is quite apathetic. Body all over extremely cold. Thermometer in rectum registers 92°.

February 17. Died.

Diagnosis during life was chronic spinal meningitis.

Summary of Symptoms.

The case ran a course of about four and a half years. The symptom first noticed was pain referred to the left heel, followed by shooting pains up or down the left side. There were also referred pains in the right side of the chest. Next came hyperæsthesia, not only cutaneous, but also muscular; for there was pain on using the muscles, and as result of the hyperæsthesia of the bladder we have frequency of micturition. There was muscular weakness and great loss of weight, and finally rigidity, which involved the muscles of respiration and led to death. These symptoms all point to thickening of the spinal membranes, with compression of the nerve-roots. They were not symmetrical on the two sides, and proceeded from below upwards. In addition there was ataxia of the upper limbs and doubtful spasticity of the lower, symptoms which imply involvement of the cord, and would be easily explained by the pathological changes actually found. No history of syphilis was obtained, but the changes in the arteries found and described below leave very little doubt as to this being the cause of the disease.

The very low temperature before death was no doubt due to deficient aeration.

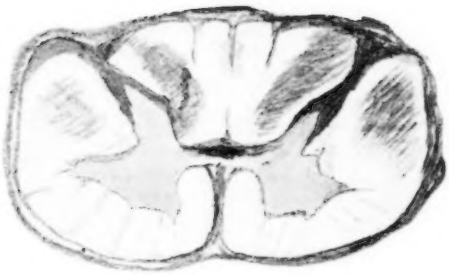

FIG. 1.
(*Semi-diagrammatic*).
Spinal Cord, Cervical region.

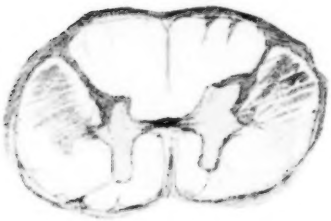

FIG. 2.
(*Semi-diagrammatic*).
Spinal Cord, Dorsal region.

FIG. 3.
(*Semi-diagrammatic*).
Spinal Cord, Lumbar region.

FIG. 4. × 50.
Periphery of Lateral Column in Cervical region.

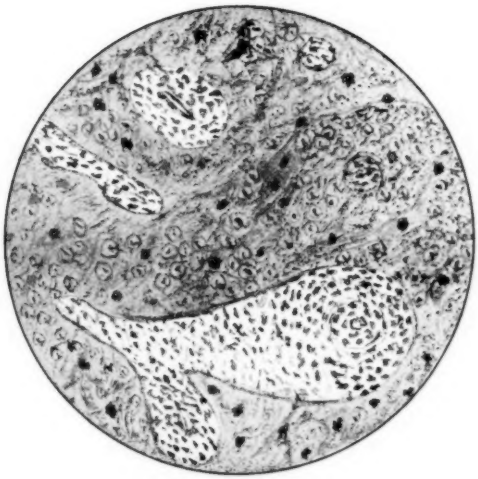

FIG. 5. × 200.
Some Arteries from Posterior Column in Cervical region.

The retraction of the head and deflection of the eyes just before death indicate some basal meningitis.

Pathology.

By the kindness of Dr. Mackie Whyte I received the brain and spinal cord.

The brain presented nothing abnormal to macroscopic examination, but as it had been in spirit, it was impossible to ascertain the presence or absence of basal meningitis. The spinal dura mater appeared quite normal, but the pia mater was tough, thick, and adherent firmly to the spinal cord, especially over the dorsal half of the circumference.

Preparations of the cord at about ten different levels showed more clearly this increase in thickness of the pia mater, and in addition sclerosis of both lateral columns throughout the length of the cord, involving both the direct cerebellar and the crossed pyramidal tracts. In the cervical region there is also sclerosis of the posterior columns, here even more marked than that of the lateral, and involving especially the postero-external tracts and the posterior root zones of Lissauer.

On close examination of the sclerosed columns, the most strikingly pathological condition is found in the arteries. These are in a condition of advanced arteritis, both endarteritis and periarteritis; the endothelial lining of the intima is proliferated until the lumen is extremely small or obliterated, while the media and adventitia are replaced by connective tissue crowded with nuclei. The arteries thus form almost solid columns, whose cross-sections take up a large proportion of the area of the section of the affected region, and contribute most to the naked-eye appearance of the sections.

These changes in the arteries are usually considered to be characteristic of syphilis; hence the title of this article.

Sections of the medulla at various levels show the pyramids along their whole length to be in the same condition as the lateral columns of the spinal cord. This is more remarkable than appears at first sight, for while regarding

the lesions in the cord alone we could assume the disease to originate in the arteries, for the districts affected are all served by the posterior spinal arteries and their branches, the anterior spinal artery and its area of supply being healthy, or but slightly affected. But when we come to the medulla we find the pyramids supplied by the anterior spinal artery are the only diseased parts, and that here the anterior spinal artery is the diseased artery, the others being healthy. This almost forces the conclusion that the origin of the disease is really in the motor tracts. As a corollary to this we might assume that the lepto-meningitis, to which the symptoms were due, was secondary to the arterial disease, and therefore to the disease of the motor tracts.

Sections of the pons showed an increase of nuclei in the coats of the arteries supplying the pyramidal fibres, but nothing approaching the condition lower down.

Sections of the cortex from the motor region showed healthy arteries and abundant healthy pyramidal cells, while tangential fibres were well developed. The nerve-cells in the gray matter of the spinal cord were healthy.

A CASE OF ERYSIPELAS OF THE SCALP WITH A REMARKABLE NERVOUS SEQUELA.

BY F. J. POYNTON, M.D.

Assistant Physician to the Hospital for Sick Children, Great Ormond Street, and Casualty Physician to St. Mary's Hospital.

THE HISTORY OF THE CASE.

THE patient, a youth, aged 17, was first an inmate of St. Mary's Hospital under Dr. Cheadle, later he was transferred to the Chelsea and Willesden Infirmaries, and finally was admitted under Sir William Gowers to the National Hospital, Queen Square.

I am indebted to the kindness of Dr. Cheadle and Sir William Gowers for leave to make use of the notes that were taken while he was under their care, and to the courtesy of the Superintendents at the two Infirmaries for information as to his condition during that period of his illness.

The duration of the illness was a long one, extending from October 20, 1897, to April 14, 1899, and it so happened that I was able to keep in touch with him throughout, and to follow his subsequent history.

E. R., aged 17, was admitted to St. Mary's Hospital upon October 20, 1897, complaining of giddiness and pain in his head and back. The illness had commenced quite suddenly three days previously, and he took to his bed the day before admission. The only fact that suggested a cause was that his mother had recently returned from a London fever hospital convalescent from an attack of scarlet fever. This raised the suspicion that the dwelling house was insanitary. He was very ill, the temperature 104°, pulse 102 per minute, respirations 30. The tongue was thickly

coated, and the bowels were constipated. Though quite sensible the least movement pained him, and he lay curled up on his side, with the eyelids closed. The aspect was pale and anxious, and the patient complained bitterly of pain in the head, in addition to which there was occasional vomiting. Some enlarged glands were detected behind the left ear, and the veins over the left side of the forehead were prominent and distended. There was no membrane upon the throat, no scarlet redness or rash upon the chest, nor did the condition of the other systems throw any light upon the condition.

Later in the day exquisite tenderness developed over the head; this was more marked upon the left side than the right, and with it there was also some tenderness down the left side of the neck. There was slight double optic neuritis, but there were no ocular palsies and the pupils reacted to light and accommodation. The tympanic membranes were quite healthy. The next day the symptoms remained much the same, except that the tenderness over the head became more general. The knee-jerks were tested several times and were never obtained. Upon October 23 a dull flush appeared upon the forehead spreading from the hair line, and the skin in this region was œdematous. The next day it was clear that he was suffering from a severe attack of erysipelas of the scalp, and from blood obtained from the spreading margin I obtained a pure culture of streptococci. Antistreptococcin in 10 cc. doses was injected upon the 26th and 27th. At this time the patient's condition was perilous. His face was now greatly swollen, the mental condition dull, and the temperature oscillating between 104° and 106°. Upon October 29 there was a crisis, the temperature fell to 99°, and desquamation commenced upon the face. After a slight relapse the temperature fell to normal upon November 4. From this date there was a steady improvement, and by the end of the month he was able to sit upon a chair, and in December was able to walk and was apparently convalescent.

In the second week of this month a curious change took place: his appetite failed and the bowels became constipated,

the temperature did not, however, rise. He became very irritable, was morose even to his friends and complained that his legs gave way suddenly letting him fall. There was no loss of consciousness. The change was so marked that he was compelled to take to his bed. Headache and vomiting now resumed, and at times he was very drowsy. The knee-jerks were now active and presented that peculiar oscillation which is seen not infrequently in chorea: there was no ankle clonus. In the middle of January there were sudden unexplained rises of temperature to 101°, the bowels remained obstinately constipated, and for the first time there was retention of urine. Both lower extremities were weak and he was unable to support his weight. The grasp was also weak on both sides. Sensation was blunted over the lower extremities, but not lost. The respiration was sighing. Pain had been complained of in the back, but it was not accurately localised, and there was no localised tenderness. Upon January 20 there was incontinence of urine. He remained in the hospital until April in much the same condition, completely bedridden, with retention varied with incontinence of urine. The lower limbs, though they could not be raised from the bed, were not spastic, nor had they wasted to any marked extent.

After leaving the hospital he was an inmate of the Willesden and Chelsea Infirmaries. Through the courtesy of the Superintendents information was obtained of his condition during this period, of which the following is a summary. He remained in very much the same helpless state, getting at first if anything rather worse, for the legs became weaker and the incontinence of urine more frequent, and in addition there was an occasional girdle pain. Towards the end of 1898 he began to improve, the legs becoming stronger.

Upon March 1, 1899, he was admitted to the National Hospital under Sir William Gowers, and for the following notes I am indebted to Dr. Edwin Bramwell, at that time the junior house physician. He was thin, his manner depressed, but intelligence good. There was some lateral nystagmus. The fingers moved well and equally, but there

was some slight weakness of the muscles of the arm and forearm upon the right side. The movements of the upper limbs were shaky and suggestive of the intention tremor of disseminated sclerosis. The handwriting was very shaky.

The abdominal muscles were weak. He was unable to raise his legs off the bed and no movement could be made at the knee or ankle joint, yet slight interrupted movements of the muscles were noticeable when movement was attempted. The toes were moved rhythmically some three to four seconds after the suggestion was made. The legs were thin; there was no rigidity. Sensation was also altered, thus there was loss of muscular sense in the right arm, and diminished sensibility to touch and pain up to the fifth rib, and analgesia to the seventh. The upper extremities were not anæsthetic. The knee-jerks were increased, but there was no ankle clonus. The plantar reflex was absent at first, but later there was a flexor response. Incontinence of urine was the rule varied by occasional retention, and the patient stated that he was sometimes aware when the urine was passed, but had no control. The urine contained pus. The bowels were constipated, but sometimes moved without his knowledge. There were no bed sores. The bladder was washed out daily, and strong faradism applied to the legs. Improvement was almost immediate. Upon March 12 there was an unaccountable rise of temperature to 103°. Upon the 18th he was so much better that the incontinence of urine had all but ceased. Upon the 21st he had complete control and could actually walk a few steps. He left the hospital upon April 14 and could then walk a mile.

After leaving the National Hospital he went to a convalescent home and returned quite strong and well. Later in the year he showed himself to Dr. Cheadle in capital health. In the autumn of 1899, he came to see me in answer to a note, and was then in robust health. His appearance had much altered, he was stout with a good colour, quite strong on his legs and with no urinary troubles at all. The knee-jerks were natural.

Thus after an illness extending from October 20, 1897, to April 14, 1899, during the greater part of which time the

patient had been bedridden, there resulted a complete recovery.

This case presents several points of interest both practical and theoretical. From the point of view of treatment, it furnishes a proof of the value of persistent and assiduous care in a condition which, on more than one occasion, looked anything but promising. If this patient had gone home early in the illness, there is little doubt that the cystitis, which even with care did eventually make its appearance, would have arisen earlier, have been more severe and would have caused irreparable damage to the kidneys.

The use of the antistreptococcic serum was rapidly followed by the termination of the acute attack, but from personal observation I do not feel convinced that this was anything but the natural crisis which would have occurred irrespective of the use of the serum.

The rapid success attending the use of strong faradism was a signal proof of the value of this method in suitably chosen conditions.

The injury to the mechanism of the bladder was another remarkable feature of this case. For a long while there was almost complete incontinence, and yet the result was recovery and complete control. The symptom-complex at the time that there was incontinence was unusual, for the lower extremities, though weak, had not wasted more than could be accounted for by disease, and the knee-jerks were more than usually active. On the other hand, when there was retention of urine there was never any rigidity of the lower limbs, nor ankle clonus. The condition was a marked contrast to that intensely organic affection of the cord, anterior poliomyelitis, in which with severe flaccid paralysis of the lower extremities the sphincters as a rule escape.

How much of this illness was functional, in the sense that it was dependent upon lack of will power on the part of the patient, is a difficult question to answer. The severe implication of the bladder certainly seemed to point to an organic lesion of some severity, and to a bad prognosis,

and even when recovery occurred, there still remained the doubt as to whether this was only temporary, and not, as has proved to be the case, permanent.

The explanation of the cause of this sequela is to a great extent a matter of hypothesis. It is not at all likely that the condition could have been produced by the injections of antitoxin, though we cannot lose sight of the fact that this serum was used upon two occasions.

The stages through which the illness passed were firstly that of the acute local infection, to which no doubt the severe cerebral symptoms at the outset were due; then a period of improvement, which almost reached the point of recovery, next a gradual mental deterioration rapidly followed by the appearance of paralytic symptoms; fourthly a prolonged stationary period, and lastly, a stage of rapid recovery.

The primary infection may have damaged the brain considerably, and the gradual development of the new train of symptoms after a latent interval resembles in this respect that occasional sequel of a brain shock or injury—chronic traumatic neurasthenia. In this case the local infection would take the place of the more gross mechanical injury.

It is also possible that this sequela was the direct result of the virus of the erysipelas which persisted in an attenuated or altered state. Much light has been thrown by neurologists upon the occurrence of nervous symptoms as an after-result, proximate or remote, of acute infections, and the question is one of far-reaching importance. Now that almost each year we are confronted with new micro-organisms, each looked upon as the specific cause of some particular disease, the complexity of the bacteriological problem is becoming very apparent. A survey of the group of coccal infections well illustrates this difficulty. There are the streptococci and the staphylococci, the diplococci of pneumonia and rheumatic fever, of scarlet fever and cerebro-spinal meningitis. There is the gonococcus and the diplococcus described by Still in posterior basic meningitis. Morphology alone can hardly be expected to distinguish them, much less any method of staining, and it seems that chemical pathology by a study of the poisons that are produced and the re-

actions that are called out will eventually afford the means of differentiating these processes.

Nervous disturbances resulting from infections are of such interest because they are to some extent a clinical expression of these subtle chemical processes. If this case be looked upon as an example of this kind then it is a fortunate one, because the organism was isolated at the commencement of the illness and the patient under observation throughout its entire course.

Reviews.

A Treatise on Mental Diseases, based upon the Lecture Course at the Johns Hopkins University, 1899, and Designed for the Use of Practitioners and Students of Medicine. By HENRY J. BERKLEY, M.D., *Clinical Professor of Psychiatry, the Johns Hopkins University, Chief Visiting Physician to the City Insane Asylum, Baltimore.* With Frontispiece, Lithographic Plates and Illustrations in the Text. (New York: D. Appleton & Company, 1900.)

THE object of the author of this treatise has been to supply English medical literature with "a comprehensive, practical work on Mental Diseases," and the production of a most admirable text-book for practitioners and students is the result. Both the wide view of the subject adopted and the absence from the work of such a chapter on the medico-legal aspects of insanity as would give it a more or less local character, fit it for the large class of readers for whom it is intended, and the general clearness of the descriptions and the interesting manner in which every branch of the subject is treated cannot be too highly praised.

The book opens with a short and lucid description of the general anatomy and histology of the cerebrum so far as this has a direct bearing on the study of mental diseases, and the absence of unnecessary details of structure has enabled the author to compress this portion of the subject into fifty pages of highly readable matter. The general structure of the cortical neurones and of the neuroglia is beautifully illustrated by drawings of Golgi preparations, and excellent diagrams of the nerve elements and the lympathic circulation of the cortex are introduced. The finer structure of the nerve-cell as shown by the Nissl method is clearly described and well illustrated, and a satisfactory account is given of the cell layers and association fibres of the cerebrum.

This part of the work is followed by a description of the general and special pathology of mental disease. In the introduction to this part the author lays especial stress on an inherited predisposition to disturbances of function and to premature retrogression of the cortical cells, the development of mental disease being associated with original defective construction of the neurone, vascular lesions occurring at the retrogressive period of life, or general toxic conditions. Whilst the description of the gross pathological anatomy of the cerebrum is short and only moderately good, that of the experimental pathology of the nerve-cell is excellent. Very clear accounts of the changes in the cell protoplasm in primary and secondary degeneration are given, as is also a description of the author's own researches on the nuclear changes due to the action of ricin and alcohol on the cortical nerve-cell, this last being of especial value owing to the relatively small amount of attention which is usually paid to this subject. The writer contrasts the excellent results obtained from experimental lesions with those in the human subject, and is inclined to lay more stress on nuclear changes than on changes in the Nissl-bodies. He also considers, from the experimental standpoint, that Golgi preparations are trustworthy as regards morbid changes, and his views in this matter, though contrary to those generally accepted, are entitled to respect in that they are founded on certain personal investigations which he briefly refers to. He, however, discounts the value of the Golgi method when applied to the human subject owing to the presence of *post-mortem* products, &c. Unfortunately the morbid changes in nerve-fibres are barely referred to, but an excellent description of the pathology of the cerebral vessels and of syphilitic vascular lesions is given. The section closes with a short illustrated description of abnormalities of the basal arteries. The illustrations in this section are only moderately good, and at least one or two cells showing Nissl changes might well have been introduced. The literature also is very incomplete, and shows an entire absence of reference to English authors, the only two writers in English referred to being Americans. This may partially explain the views of the author on the subject of cell changes in the human subject. The same remark with reference to the literature might be made in the case of several succeeding sections of the work.

In the first group of mental disorders treated, namely, "the idiopathic insanities without ascertainable alteration of the brain substance," is included a description, occupying only three pages

of letterpress, of secondary dementia. It is to say the least somewhat remarkable that a group which includes probably half the total inmates of asylums should be dealt with so very briefly, and also that a condition which in its later stages presents gross pathological appearances second in degree to those of general paralysis alone, should be placed under the heading of idiopathic insanity without ascertainable pathology. This contradiction is emphasised by the presence in the article of a cut illustrating marked dilatation of the lateral ventricles in a case of chronic mania, which, however, is not referred to in the text.

Under group 2 are included the insanities consecutive to demonstrable organic lesions. The account begins with a description of "progressive paralysis of the insane." The author lays the usual stress on syphilis as the pre-eminent ætiological factor in the disease, and it is satisfactory to note that he includes heredity as the other important predisposing cause, here agreeing with Näcke, who found a hereditary history of insanity in 50 per cent. of his cases. The symptomatology of the disease is good and is followed by an excellent summary of the more important facts. The author refers shortly both here and in a later chapter to the juvenile form of the disease. In the account of the pathology of general paralysis the writer accepts the theory that the disease is of vascular origin and usually caused by the direct or indirect effects of syphilis on the cortical vessels, and considers the changes in the nerve-cells to be secondary and indefinite. "The Golgi and Nissl methods have unfortunately added but little to our knowledge of the conditions of the neurone in paresis." The description of the pathology of the disease whilst as a whole good rather partakes of the nature of an *ex parte* statement of facts, and in this agrees with the author's general tendency to ascribe the majority of the insanities of this group to vascular lesions of primary origin, or due to the action of toxines, with secondary neuronic changes. This is curious when one considers the high metabolism and the unstable nature of the neurone, and the fact that, except in senile cases, the author acknowledges the influence of heredity in inducing defective construction and imperfect vitality of this structure. In the article on syphilitic insanity the author makes the rather surprising statement that this disease "is quite as frequent or even more common than general paresis and tabes dorsalis," and then explains this by stating that "all cases under 40 years of age that are hemiplegic or monoplegic, with temporary or permanent mental disturbance, and in whom there are no indications of

renal or cardiac lesion, are with us ascribed to syphilis, as are likewise many of the obscure dementias that occur between the 25th and 40th years." The pathology of syphilitic insanity is described in half-a-dozen lines, which is very unsatisfactory in view of the above statement. In the article "psychoses of old age," quite a lengthy description, occupying nine pages, is given of the pathology of the disease and especially of senile vascular changes in the cerebral vessels, and this is followed by a short account of organic dementia. The clinical description of the alcoholic insanities is particularly good, but the pathology given consists almost entirely of an account of the author's researches on alcohol poisoning in rabbits.

In the fourth section, idiocy, cretinism and imbecility are treated in an article which, both clinically and pathologically, is much above the average, and in the description of imbecility the author refers to the frequency of insanity, which often ends rapidly in dementia, in the higher class of imbeciles. In this section is included an excellent article on cranial measurements and the stigmata of degeneration, with an illustrated description of the author's observations on abnormalities of the palate.

Whilst the general descriptions in the clinical section are good it is difficult to see why groups 1 and 3 are separated from one another, and it is remarkable that no reference, even under another name, is made to hypochondriasis, which is such a common symptom-complex in both the sane and the insane. The clinical section would probably have been improved by an introduction dealing with those details of psychology which have a special bearing on the phenomena of mental disease, and with the symptoms of mental derangement considered from a psychological standpoint, for as the subject is treated in the present work the student will necessarily gain the impression that insanity is a series of diseases instead of a series of symptom-complexes which have a common origin in lesions of those parts of the nervous system concerned with the processes of thought.

In text-books written by such an acknowledged authority as the author of this work, it is no doubt desirable that the individuality and opinions of the writer should have all due attention paid to them. It seems a pity, however, that in so many works, as in the present one, so much stress should be laid on individual opinion, and so little attention paid to the views of other authorities, for a text-book should be essentially a statement of known facts and theories rather than a collected edition of the researches and views of the author.

The index is excellent, and the illustrations as a whole are very good, especially the photographs of patients. Here and there words unfamiliar to the English reader such as "obstipation," "arrestation" and "coprolalia" are to be met with, and occasionally, according to American custom, the words are curiously divided, as for example, "cellu-lifugally," "ma-niac," and "arterioscle-rosis."

The book as a whole, apart from the blemishes to which attention has been drawn, is an excellent one, and should not only be most useful to the wide class of readers for whose use it is written, but will also be found to contain a considerable amount of matter of interest to the alienist.

J. S. Bolton.

A Text-Book of Pathology in Relation to Mental Diseases. By W. Ford Robertson, M.D., *Pathologist to the Scottish Asylums; formerly Pathologist to the Royal Edinburgh Asylum.* Illustrated with 16 lithographic plates in black and in colours, and 13 engravings. (Edinburgh: William F. Clay, 18, Teviot Place, 1900.)

It is doubtful whether the publication of a work of this kind is desirable in the present early and rapidly-advancing state of our knowledge of the pathology of the cortical neurone, as such a text-book is necessarily almost out of date by the time it reaches the hands of its readers. Few neuro-pathologists, however, are better qualified for such a task than the author of the present work, for, as he states in the Preface, he has devoted the past seven years to researches in this subject with a definite view to their final publication in book form. The work is consequently to a large extent, and especially in the case of some of the more valuable chapters, a republication of the author's previous papers. Of the twenty-nine plates, for example, which the work contains, only eight are new, the remaining twenty-one having been previously published by the author either alone or in conjunction with others. This fact, whilst not in any sense decreasing the value of the book, tends to throw into undue prominence the special researches which have been carried out by the author.

It is impossible to too highly praise the chapters on hæmatoma auris, the morbid conditions of the dura mater and pia-arachnoid,

and the structure and morbid conditions of the neuroglia. In the chapter on hæmatoma auris the literature is fully referred to, and a well-balanced criticism is followed by an excellent summary of conclusions. In that on the dura mater the minute description of the degenerative conditions of this membrane and their relation to the formation of sub-dural false membranes is especially good, although the numerous illustrations are very diagrammatic, and do not reach the high standard of the description. In the excellent account of the normal structure of the pia-arachnoid, the author lays stress on the virtual absence of capillaries from this membrane, which is a fact hitherto not generally recognised. The chapter on the intracranial blood-vessels and the special histological characteristics of the intracerebral capillaries is also good. That on the neuroglia is, however, probably the most valuable chapter in the book, and contains the first complete account of the author's researches on this subject. He strongly supports the view recently formulated by certain authors, and especially by Capobianco and Fragnito from their rearches on the developing chick, that part of the neuroglia is mesoblastic in origin. The special characters of the two varieties of neuroglial cells are minutely described. Those of epiblastic origin or "neuroglia" are "special highly-branched cells, which vary greatly in size, and in the number and arrangement of their processes. . . . Their protoplasm . . . has become in greater part differentiated into a denser substance, which forms fine fibres. . . . Many of them are attached to the adventitia of the vessels either directly or by means of fan-shaped expansions." The cells of mesoblastic origin, or "mesoglia," are "probably about equally numerous, . . . have processes which are composed of the same substance as the cell body, and are generally markedly dendritic. Moreover, they have no connections with the vessels or other structures."

The chapter on the morbid conditions of the nerve-cell is mainly an amplification of the author's previously published critical digest. Whilst it is an excellent account of our present knowledge of the general pathology of the nerve-cell, a more minute description of the various types of primary cell degeneration might have been introduced, and the illustrations on plates xxvii. and xxviii. are not only unsatisfactory, but not a single typical example of a cell undergoing acute degeneration is shown. There is also no adequate description of or discussion on the morbid conditions of medullated nerve-fibres.

In chapter XIV. is given a brief account of the pathology of

certain of the clinical types of mental disease. The author throughout inclines to accept the toxic or auto-toxic theory of the pathogenesis of the insanities (excluding imbecility) and lays relatively little stress on hereditary influences. He criticises Mott's advocacy of the theory of Tuczek that general paralysis is a primary degeneration of the nervous elements of the cerebral cortex, and accepts the toxic theory of Angiolella, giving an excellent description of the lesions of the cerebral capillaries in this disease. He does not however satisfactorily show that these lesions may not equally result from toxins derived from neuronic degeneration, and finds it "difficult to understand why the capillaries of the frontal and central convolutions should be specially liable to involvement." In his explanation of this he omits any reference to the anatomical arrangement of the veins of the fronto-parietal region, on which stress is laid by Mott, and to the fact that this region is the part of the brain most affected in ordinary insanity.

Chapter XII., on the cerebral circulation, is the least satisfactory part of the book. The author in it severely criticises the researches of Leonard Hill on the physiology and pathology of the cerebral circulation. In his attempted refutation of the Monro-Kellie doctrine of the incompressibility of the brain, he confuses compressibility with elasticity (pp. 293-4), and as a result denies Hill's statement that "the volume of the blood in the brain is in all physiological conditions but slightly variable." He then shows how, if Hill's facts be *not* accepted, the mechanism of the intracranial circulation can be explained by alterations in the amount of lymph present in the intracerebral lymphatics. He also accepts Cavazzani's statement (1892) that the cervical sympathetic contains vaso-constrictor and vaso-dilator nerves for the brain, in preference to those of Hill (1896 and 1899) that "evidence is not forthcoming of the existence of any local vaso-motor mechanism," and that "the cerebral circulation is controlled by the vaso-motor centre acting on the splanchnic area." In view of the author's denial of the Monro-Kellie doctrine it is not surprising to find that true œdema of the brain depends on conditions "the exact nature of which must still be regarded as obscure." Whilst the opinions of the author on this subject are entitled to consideration it is probable that readers will consider this chapter to be unsatisfactory, and it is to be hoped that it will be modified in a future edition of the work.

In minor details the book presents shortcomings of less importance. In the chapter on histological methods, for example,

whilst the methods or modifications of methods preferred by or originated by the author are fully described, no description of the Weigert-Pal and Nissl methods is given. It is also to be regretted that the subjects which have not received the special attention of the author are so briefly referred to. Taken as a whole, however, the book is well written, the general arrangement is satisfactory, and the index, though not copious, is good. The work is a valuable contribution to neuro-pathology and will be of considerable assistance to the original workers for whose use it is intended by the author.

J. S. BOLTON.

Die Vasomotorisch-trophischen Neurosen. Dr. RICHARD CASSIRER. Karger, Berlin, 1901.

UNDER this title Dr. Cassirer, in a monograph of 600 pages, gives us an exhaustive study of such diseases as acroparæsthesia, erythromelalgia, Raynaud's disease, scleroderma, angioneurotic œdema and multiple gangrene.

As a fitting introduction to the work the first chapter of 100 pages is devoted to the physiology and anatomy of the various nerve-centres and tracts directly associated with the vasomotor, secretory and trophic functions of the nervous system. The several diseases are then considered separately in the following chapters.

Dr. Cassirer, in addition to his own observations made at Professor Oppenheim's Nerve Clinic in Berlin, has collected together a vast amount of information from other sources, and consequently he is able to tell us nearly everything of importance that is known of the ætiology, symptoms, pathology and treatment of these diseases.

The chapter on Raynaud's disease in particular is most complete and speaks to the thorough way in which the author has set about his laborious task.

Throughout the work numerous cases are considered in detail in order to show how intimately these diseases are related, and in the last chapter a brief summary is given of the author's conclusions, together with a suggestive table which shows the leading symptoms of these neuroses.

Vasomotor disturbances, as represented by local syncope, asphyxia and hyperæmia, are the essential changes which underlie all these conditions, and around these are grouped in various ways sensory, secretory and trophic disturbances.

The marked prominence of any one of these local disturbances changes the clinical type of the disease and thus produces a clinical variety which stands intermediate between one disease and another.

Raynaud's disease, on account of its multiplicity of symptoms, is placed as the centre around which the other diseases are grouped.

In this disease vasomotor, sensory, secretory and trophic disturbances are all represented in varying degree in different cases.

If, however, these disturbances become more marked in one direction we have a change in the clinical picture.

A case of Raynaud's disease may, for instance, show trophic changes in the fingers and toes identical with those seen in sclerodactylie.

Sclerodactylie again is sometimes associated with ordinary diffuse or circumscribed scleroderma, so that we have a clinical series of cases starting from Raynaud's disease and ending in scleroderma.

Again, if hyperæmia is a marked symptom in Raynaud's disease and other symptoms stand in the background we approach the condition of erythromelalgia.

In the same way the prominence of gangrene causes the clinical picture to approach that of multiple gangrene. Such examples briefly show the direction in which Dr. Cassirer formulates his conclusions.

An extensive bibliography, so necessary in these days, helps to complete a work which cannot fail to be an important book of reference to those who may be studying this interesting group of diseases.

E. Stainer.

PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF LONDON.

AT the CLINICAL MEETING held at 11, Chandos Street, on Thursday, March 21, at 8.30 p.m., the following cases were shown :—

(I.) Dr. F. E. BATTEN.—*Four cases of congenital ataxia in children.*

(1) F. W., aged 5, was first seen in July, 1900. He was the third of three children, of whom the first two were quite healthy. The labour was natural but the mother was not in good health while carrying the child. He learnt to talk at a year old. The nystagmus which is present has been noted since birth, and the squint a few months after birth. He has never learnt to walk.

When attempting to stand or walk, the child was very ataxic; there was marked incoordination of the hands, constant nystagmus, and the articulation was very defective. There was no rigidity of the legs, and no pes cavus or spinal curvature. The knee-jerks were active, there was no ankle clonus, and the plantar reflex gave a flexor response.

The boy was clean in his habits, played with his toy, said little pieces of poetry, and was contented.

His present condition (8 months later) has much improved. He can now walk, but is somewhat ataxic. There is less incoordination of his hands, but his articulation is still very defective.

The knee-jerks are present, there is no ankle clonus, but the plantar gives an extensor response.

No satisfactory view could be obtained of the discs.

(2) D. S. This child was shown at the Society one year ago. She is now 5 years old. She is the first and only child, and instruments were used at her birth. When last shown she pre-

sented a markedly incoordinate gait, incoordination of the arms, and difficulty in articulation. There was considerable difficulty in swallowing. The knee-jerks were active. She has improved a good deal in her walking but is still very ataxic.

(3) R. C., aged 6, was first seen in July, 1900. She was the sixth of seven children, all the others being healthy. The labour was prolonged, and was followed by a flooding. She began to walk when 4 years old, but tumbled about a good deal. She learnt to talk at 2 years, and has always been sharp, intelligent and clean in her habits. She is very slow in eating, and has difficulty in swallowing.

When first seen she walked in a very incoordinate manner, and was very liable to fall backwards. There is more incoordination in the right arm and leg than the left. There is no rigidity. The speech is slow and drawling, and not easy to understand. There is no nystagmus and the discs are normal.

The knee-jerks are active; there is no ankle clonus, and the plantar reflexes give flexor responses.

(4) L. T. This child was first seen in 1894, when 1½ years old, and she was in the Hospital for Sick Children under the care of Dr. Lees, with shaking of the limbs, inability to sit up, inability to talk, and nystagmus.

She was seen again in 1898, when 5 years old. She could then talk, but could not walk; there was much tremor of both hands and rotatory nystagmus.

The legs were rigid and ankle clonus was present. The intelligence was good, and the child was clean in her habits.

(II.) Dr. COLMAN.—*Case of* (?) "*mind blindness.*"

Edwin C., aged 56. In September, 1900, had some kind of fit, after which he was "delirious" and did not recognise his surroundings. He recovered his ordinary state of health in a fortnight, but was found to be blind.

He is unable to distinguish objects well, there being evidently much dulling of ordinary light-perception; but he can count fingers and can make out the shape of objects that are shown him (*e.g.*, he says that a watch is round, has lines starting out from the centre and marks round its edge, but that he has no notion whatever what the object is. If he hears it or touches it he recognises it at once).

He will fail to recognise that an object is a book, but once he has assured himself by touch that it is a book, he will be able to

pick out letters or even simple words, showing that he is not completely blind. He stumbles over obstacles, however, and has to be led about. His fundus is normal, and his pupils react quickly to light.

He has marked loss of memory for recent events, but there is little intellectual impairment beyond this.

In January he had another fit. There was no loss of consciousness. He was word-deaf for some hours, and had "gibberish aphasia," but next day there was no trace of aphasia.

(III.) Dr. WILFRED HARRIS.—*Case of syringomyelia following infantile paralysis.*

E. C., a married woman, aged 42, when aged 3 was stricken with infantile paralysis, damaging the left arm and leg and leaving, as permanent effects, left talipes equinovarus due to complete absence of the tibialis anticus and weakness of the extensor communis digitorum; in the left upper extremity some general smallness of the arm and forearm muscles with slight comparative weakness, and complete atrophy of the thenar eminence, and partial of the first dorsal interosseus, and general smallness of the bones of the hand. Beyond permanent chilliness of the left hand and some awkwardness in holding things, she suffered no disability until twelve months ago. For fourteen years she was in domestic service, and she afterwards married and had children. About March, 1900, she first noticed weakness in raising the left arm, and almost constantly since has complained of pain in the left shoulder and hand, and more recently in the spine and elbow. When first seen, last October, there was, in addition to the condition above described, marked weakness and some atrophy of the left deltoid, biceps, supinator longus supra and infra-spinati, and rhomboids, with fibrillary tremors in the deltoid and biceps. Partial tactile anæsthesia and analgesia above left elbow, extending over shoulder on to chest, back, and neck up to the mid-line and down to the xiphisternum. No scoliosis. Knee-jerks equal and brisk. Bilateral extensor plantar reflex. Jaw-jerk brisk. No bulbar symptoms nor nystagmus nor contraction of the visual fields. In January she complained much of pain in the cervical spine and elbow, but no spinal tenderness on pressure. She now volunteered that she could hold anything hot in the left hand without feeling it, though she is certain that the sensation of the hand was normal until recently. Since January the tactile anæsthesia and analgesia have invaded the whole of the left half of the body, head, limbs,

and trunk, though the analgesia is most marked now on the left hand and wrist, and is also present on the right hand and lower part of forearm. The left knee-jerk is now much brisker than the right, and her left arm and leg have become much weaker lately. The electrical reactions show no reaction of degeneration in the deltoid, biceps, or supinator longus, but diminution of contraction and slight sluggishness to both forms of current.

(IV.) Dr. FARQUHAR BUZZARD.—*Atrophy of arm with anæsthesia over distribution of first dorsal root.*

A. O., widow, aged 40, under the care of Dr. Beevor, as an out-patient at the National Hospital, Queen Square.

Five years ago, whilst convalescent from Malta fever, the patient woke up one morning to find that she had lost power in her right hand, and that she had pain shooting down her right arm from the region of the neck and shoulder. The pain has recurred at intervals, and the loss of power has remained; three years later wasting of the hand muscles commenced, and has quickly progressed. At the present time, in an otherwise healthy woman, there is marked atrophy and palsy of the intrinsic thumb muscles of the right hand, some weakness of the interossei and of the long flexors of the fingers, complete analgesia and therm-anæsthesia with impaired sensibility to touch over an area on the inner surface of the arm corresponding to the distribution of the first dorsal root.

(V.) Dr. RISIEN RUSSELL.—*Syringomyelia with "Charcot joint."*

J. C., male, aged 29 years. History of old trophic lesions and burn without pain on left hand. Weakness of left arm for three months, chiefly affecting abduction. Muscles unusually well developed, and none of them atrophied. "Charcot joint" left shoulder. Nystagmus on turning eyes to either side. Slight right facial weakness. Thermal anæsthesia left side of face, areas on trunk and both arms, analgesia and some tactile anæsthesia on left arm. Cold test tube causes feeling of burning left arm. Very slight lateral curvature of spine. Knee-jerks present. No ankle clonus.

(VI.) Dr. W. A. MARRIS.—*Cases resembling disseminated sclerosis.*

(1) J. B., male, aged 18, under the care of Dr. Ferrier. Family history is good. Patient was normal till three years

old, when he had scarlet fever. After this he was unable to walk and trembled all over. In three months he learnt to walk again, but the trembling is still present. The disease is not progressive. Patient's physical and mental development are very backward. The speech is thick and slow. The cranial nerves are normal, there is no nystagmus and no optic atrophy. The chief symptoms are the tremor of limbs and body, and slight spasticity in the legs. The tremor occurs on movement, disappears on resting, but it is not of crescendo type, and there is no ataxy in the arms. There are no sensory changes. The deep reflexes are increased, and the plantar reflexes are of extensor type. Patient has lately learnt to control his sphincters. Double pes cavus is well marked.

(2) H. W., male, aged 34, under the care of Sir W. Gowers. Patient's uncle had religious mania late in life, and a brother of the patient is suffering like the patient himself; otherwise the family history is good.

Patient was noticed to walk badly when twelve years old, and the illness has steadily progressed since. He has had no other illness. Mentally he is deficient, and his speech is markedly affected.

The optic discs are normal, there is no nystagmus, and the pupils react in every way. Slight facial weakness on the left side, and there is a very set expression of the face. The upper limbs show a fine tremor, but no definite ataxy or intention tremor. The legs are rigidly extended, and there is much spontaneous rectus-clonus. The plantar reflexes are extensor in type, the sphincters are unaffected. Pes cavus is present on both sides.

(3) C. W., brother of Case 2, aged 22, under the care of Sir William Gowers. Patient had pneumonia when 1 year old, and no illness since. Was later than the rest of the family in learning to walk and never strong on his legs. At 10 years of age difficulty in walking was distinct, and has slowly become worse. At present mental state is good, speech is slow and awkward. There is no pallor of the discs, no nystagmus, nor other affection of the cranial nerves. The upper limbs are normal, without any tremor or ataxy. The lower limbs are stiff, but all movements can be performed except dorsiflexion of the ankles. His gait is unsteady, and the feet drag. On the left side the toes alone reach the ground. There is slight patellar clonus, but no ankle clonus. The plantar reflexes are of extensor type. The sphincters are

unaffected. Pes cavus is marked on both sides, and there is considerable kyphosis in the dorsal region.

(VII.) Dr. STILL.—*A case of paramyoclonus multiplex.*

Alfred R., aged 8½ years. His mother says he had "the jumps" in all his limbs for two years; the affection has varied in degree from time to time, but is worse the last few months.

No cause is assigned for its onset. He has had no previous illness, except measles and whooping-cough some years ago. No epileptiform attacks at any time.

Mother has had no miscarriages; patient is the first child, one other living and healthy. Father died with general paralysis three years ago.

Boy is fairly nourished; rather slow, but intelligent; speech deliberate and rather monotonous, with tendency to space words.

Sudden contractions of individual muscles, or portion of a muscle, not sufficient to cause movement of limbs, are seen, chiefly in the thighs where the quadriceps extensor of each side shows sometimes a single twitch, sometimes a short series of clonic contractions; the deltoid, triceps, biceps and scapular muscles are similarly affected, and, less often, the muscles of the leg, forearm and hand; similar twitchings are sometimes well marked in the rectus abdominis and the muscles of the back, and also in those of the nose and lips. The tongue also shows sudden irregular quiverings and twitchings, probably similar in nature to those elsewhere. A sudden sniffing sound, or slight grunt, which occurs occasionally, appears to be due to irregular contractions of respiratory muscles.

The affection appears to be chiefly symmetrical, but the movements on the two sides are not always isochronous. Voluntary movements seem to diminish the abnormal contractions, which are most noticeable when the child lies quietly in bed; they seem, however, to be absent in sleep.

Gait is normal, except that rarely the boy is pulled up suddenly by straightening of the knee owing to sudden contraction of the quadriceps femoris.

Electrical reactions are normal; sensation normal.

Tendon-jerks all very brisk; plantar reflexes normal (flexion).